

62310600R



NLM 05093304 3

NATIONAL LIBRARY OF MEDICINE



and Welfare, Public



and Welfare, Public



and Welfare, Public



and Welfare, Public



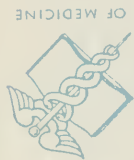
and Welfare, Public



and Welfare, Public



Health, Education,



Health Service



Health, Education,



Health Service



Health, Education,



Health Service



US Department of



Bethesda, Md



US Department of



Bethesda, Md



US Department of



Bethesda, Md



Bethesda, Md



US Department of



Bethesda, Md



US Department of



Bethesda, Md



US Department of



Health Service



Health, Education,



Health Service



Health, Education,



Health Service



Health, Education,



and Welfare, Public



and Welfare, Public



and Welfare, Public



and Welfare, Public



and Welfare, Public



and Welfare, Public



Health, Education,



Health Service



Health, Education,



Health Service



Health, Education,



Health Service



US Department of



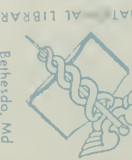
Bethesda, Md



US Department of



Bethesda, Md



US Department of



Bethesda, Md



Bethesda, Md



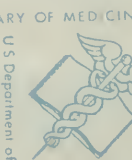
US Department of



Bethesda, Md



US Department of



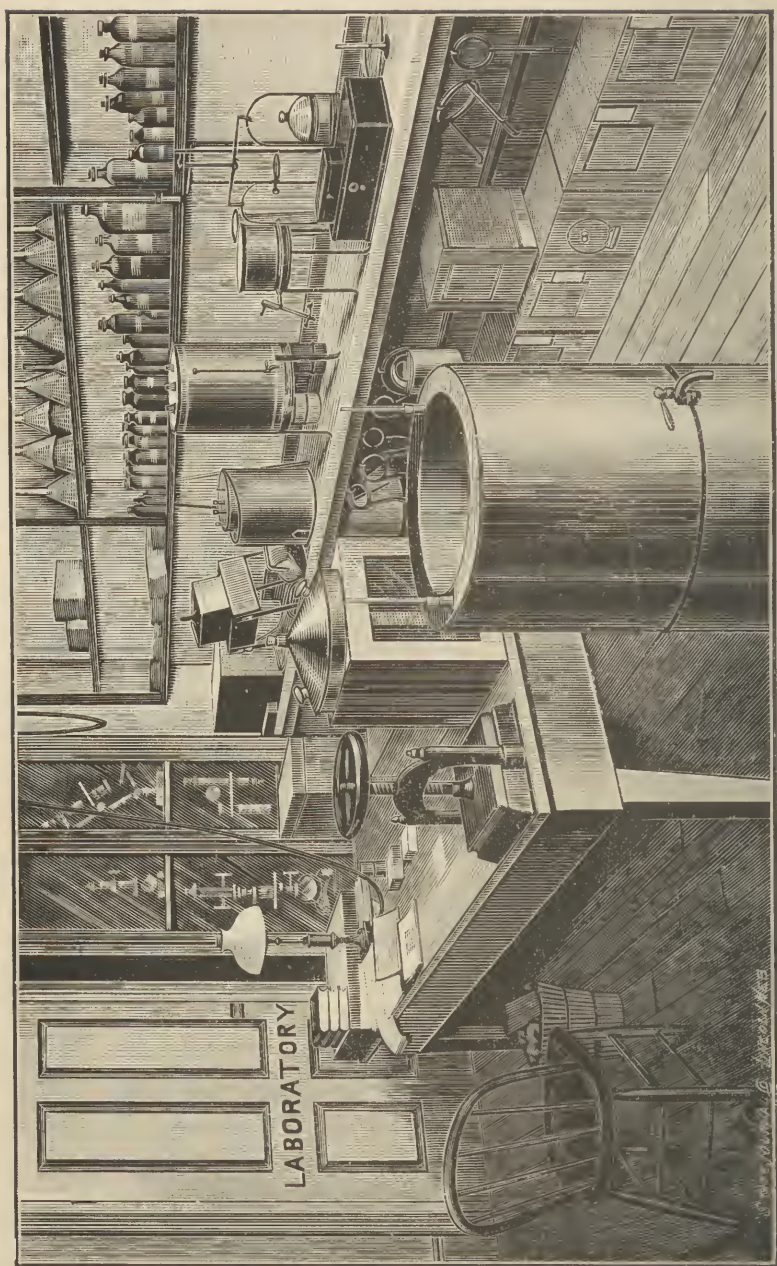
Bethesda, Md



US Department of







al

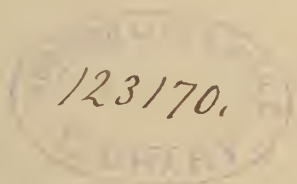
SWINE PLAGUE,

WITH ESPECIAL REFERENCE TO THE PORCINE PESTS OF THE WORLD. AN
ETIOLOGICAL, PATHO-ANATOMICAL, PROPHYLACTIC, AND
CRITICAL CONTRIBUTION TO

GENERAL PATHOLOGY

AND

STATE MEDICINE



BY

FRANK S. BILLINGS, ✓

DIRECTOR OF THE PATHO-BIOLOGICAL LABORATORY OF THE STATE UNIVERSITY
OF NEBRASKA.

LINCOLN, NEB.:
JOURNAL COMPANY, STATE PRINTERS.
1888.

To the Honorable Members of the

STATE BOARD OF AGRICULTURE,

To whose suggestion the inauguration of this work is due, and to the
Honorable

BOARD OF REGENTS,

Of the State University of Nebraska, whose cordial adoption and support of the recommendation of the representative live stock men of the State have made possible the results thus far achieved, this second report is most respectfully and gratefully dedicated by

THE AUTHOR.

CONTENTS.

	PAGE
PART I.—HISTORY, GEOGRAPHICAL DESCRIPTION, ETIOLOGY.....	7
History of swine fever in England.....	9
Causes of swine plague.....	12
Internal causes.....	13
External causes.....	17
Diseased swine as a cause of swine plague.....	18
Earth, pens, infected refuse as causes of swine plague.....	19
Proof that swine plague is an extra-organismal septicæmia and not a contagious	23
More evidence that the land and refuse are among the causes of swine plague..	28
Influences of seasons, heat, cold, rain, and moist soils upon swine plague.....	29
Influences of running streams upon the extension of swine plague.....	31
Rats as a cause of extending swine plague.....	33
Nature of swine plague.....	36
Salmon's micrococcus period in the etiology of swine plague	38
Salmon "stamps out" Dr. Detmers.....	39
Some contradictions by Salmon.....	40
Investigations of swine plague—Salmon.....	43
Salmon discovers a "new microbe" as the cause of swine plague.....	52
"The bacterium of swine plague"—1885—Salmon.....	54
"Conclusions of the investigations concerning the cause of American swine plague"—Salmon.....	54
"Conclusions"—Salmon	60
Comparison of Mr. Salmon's assertions as to the cause of swine plague from 1880-1885.....	61
"Two different germs found in hog cholera"—Salmon.....	64
Comparative resume of Mr. Salmon's views upon the swine plague.....	73
Further evidence.....	84
Other contradictions by Salmon.....	85
Further evidence of Mr. Salmon's unreliability.....	87
Mr. Salmon's experimental evidence that he never saw the germ of swine plague.....	88
Summary of Salmon's contradictions.....	92
THE SPECIFIC AND ONLY GERM OF SWINE PLAGUE.....	93
Dr. H. J. Detmers, the first discoverer of the germ of swine plague.....	96
Personal observation upon the germ of swine plague.....	102
Most practical methods of obtaining pure cultivations from the tissues of dis- eased animals.....	102
Morpho-biological characteristics of the germ of swine plague.....	104
Deportment of the germ of swine plague in beef infusion gelatine.....	114
Is the bacterium of swine plague motile or not?.....	115

	PAGE
PART II.—PATHOLOGICAL ANATOMY IN SWINE PLAGUE.....	117
Necroscopical observations upon hogs diseased with the swine plague.....	119
Necroscopical notes from inoculated hogs.....	153
Swine plague in Great Britain and Europe.....	165
Swine plague in Great Britain.....	165
Symptoms and course.....	173
Constitutional symptoms.....	173
Local external lesions.....	174
Diagnosis.....	176
Pathological anatomy.....	177
Gastro-intestinal lesions.....	180
Glandular lesions, lymphatic and mesenteric.....	183
Treatment.....	184
Prevention and suppression.....	184
Swine plague in Europe.....	186
The "Wild und Rinder-seuche".....	188
Loeffler's evidence.....	190
Professor Schutz's evidence.....	199
Schutz's cases of swine plague.....	204
Lesions described by Roloff show that the genuine swine plague exists in Germany, and when taken in connection with those described in the second section of Schutz's work complete the full picture of the disease.....	211
"Scrofulous cascous enteritis".....	211
"Necroscopical observations".....	211
Swine plague in Sweden and Denmark.....	217
Swine plague in France—Cornil-Chantemesse.....	220
Swine plague in France—Rietsch and colleagues.....	222
SALMON'S HOG CHOLERA.....	226
Are there two swine plagues known in the United States at present?.....	226
Investigations of swine diseases—Salmon.....	227
Nature of the disease—Salmon.....	234
The bacterium of swine plague—1885—Salmon.....	237
Some examples of Mr. Salmon's inconsistencies and contradictions with reference to his germs of hog cholera and the swine plague.....	239
How Mr. Salmon's "hog cholera microbe" differs from that of swine plague in manner of action.....	241
"Dr. Salmon's swine plague bacteria of 1885"—Detmers.....	247
Evidence that there is but one swine plague in the United States by other American observers.....	251
Letter of Dr. Detmers to the author.....	255
PART III.—NATURE, SYMPTOMS, DIAGNOSIS, PREVENTION.....	261
Nature of swine plague.....	261
How and where did or does the cause of swine plague originate.....	267
Contagious or infectious.....	267
An extra-organismal infection and not a contagion.....	269
Swine plague, then, is an infectious disease.....	279
Malarial-infectious diseases.....	282

	PAGE
Nature of swine plague determined by the intra-organismal action of the inficiens	282
Swine plague a septicæmia	282
Are these losses the result of a single disease?—Salmon.....	283
Authoritative evidence that swine plague is a septicæmia	286
Swine plague a septicæmia	296
Swine plague an extra-organismal septicæmia.....	298
Swine plague a septicæmia, as shown by its lesions.....	299
Contagious diseases more specific in their lesions than infectious.....	300
The action of the bacteria in swine plague.....	302
The pulmonary lesions in swine plague.....	303
Genesis of destructive pneumonia in swine plague.....	304
The intestinal lesions in swine plague	307
Can swine plague be induced by feeding?	307
Feeding experiments in rabbits	308
Feeding experiments in hogs	309
Genesis of the intestinal lesions	315
What then is the swine plague?.....	318
INTRA-VITAL PHENOMENA IN SWINE PLAGUE.....	320
The action of the bacteria in swine plague	322
DIFFERENTIAL DIAGNOSIS.....	330
What constitutes identity in germ diseases.....	330
Morpho-biological resemblances of micro-etiological organisms not sufficient evidence upon which to assert that the diseases from which such organ- isms are derived are necessarily one and the same disease.....	330
PORCINE ERYSIPELAS.	
Rothlauf—Rouget	338
Clinical phenomena in Rothlauf	339
Post-mortal phenomena in Rothlauf.....	340
Diagnosis in swine plague.....	342
PROPHYLAXIS	343
The prevention of swine plague.....	343
The preservation of the public health and protection and augmentation of the public wealth.....	345
The support and control of scientific research is the only foundation upon which to build and develop the above.....	349
An appeal for a national laboratory	349
It must be under the U. S. marine hospital service	350
The hygienic prevention of swine plague.....	353
Resume	362
THE REGULATIVE PREVENTION OF SWINE PLAGUE BY THE STATE	363
When swine plague exists in adjoining states	363
Swine plague introduced in transit.....	365
When the disease exists in the state.....	366
Disposal of sick and dead hogs.....	368
State remuneration for hogs killed by the authorities	369
THE PREVENTION OF INFECTIOUS SEPTICÆMIÆ BY INOCULATION.....	371
Empysema infectiosum—Black leg—prevented by inoculation	378

	PAGE
Evidence which shows that inoculation must be eventually achieved in swine plague.....	382
Table of experiments, 1886	384
Test experiment in the field, 1886.....	387
Test experiments with a virulent material.....	388
Salmon on the prevention of swine plague by inoculation	389
More experimental proof that inoculation will prevent swine plague.	396
Illustrations	402

PART I.

LITERATURE, HISTORY, GEOGRAPHICAL DISTRIBUTION, AND ETIOLOGY OF SWINE PLAGUE.

LITERATURE, HISTORY, GEOGRAPHICAL DISTRIBUTION, AND ETIOLOGY OF SWINE PLAGUE.

DEFINITION.

The swine plague is a specific infectious disease, peculiar to swine only, which finds its idiopathic origin in a specific micro-organism belonging to the ovoid-belted group of germs. This organism finds its source of primary and original development in the earth and earthy or refuse material, and then the swine become infected, and again infect the same kind of materials through their excreter, or it is done through the cadavers of dead swine. The primary disease induced in swine is a blood poison by means of the secretion of specific germs; hence swine plague is a septicæmia, and, because the germ finds its primary origin outside of the porcine organism, it is an extra-organismal-septicæmia. It is not contagious. The secondary lesions are pneumonia and those of the large intestine, though broncho-pneumonia and a certain class of intestinal lesions may occur at about the same time as the strictly septicæmic when the infecting organisms gain entrance to the infected, by means of either of these tracts.

HISTORY AND GEOGRAPHICAL DISTRIBUTION.

It seems that we know less of the history and geographical distribution of the swine plagues of the world than of almost any other contagious or infectious disease of animal life. As to the geographical distribution, we know that the swine plague proper has a very extended dispersion over the temperate parts of the continent of North

Literature: Reports of the United States Agricultural Department, 1878 to date. Veterinary Journal, London, England, Vols. 2, 5, and 6. Third Annual Report of the National Veterinary Association of Great Britain. Report upon Swine Fever in Great Britain, Privy Council Office, 1886. Klein, Micro-organisms and Disease, 1885. Comptes Rendus, 1887-88. *Lerbuch der Specieellen Pathologie der Haushiere*, Friedberger-Froehner, Vol. II., 1887. *Arbeiten, a. d. Kaiserlichen Gesundheits Ampte*, Berlin, 1886. Roloff, *Die Schwindzucht, fettige Degeneration, Scrofulose- und Tuberculose bei Schweinen*, 1875. *Centralblatt fuer Bacteriologie und Parasitenkunde*, Vol. III. *Wochenschrift fuer Thierheilkunde* (Adam's), 1887, 1888. Fifth Annual Report of the Ohio Agricultural Experiment Station, 1886. American Veterinary Review, April, 1888. *Journal of Comparative Medicine*, April, 1888. *Rothlauf der Schweine*.—Lydtin and Schottelies, 1885. *Virchow's Archiv*, Vol. 95, 1885. *Fortschritte der Medicin*, Vol. 6, 1888. *Aristotle's Thierkunde*.—Aubert und Weiner, 1868. Billings, *Relation of Animal Diseases to the Public Health*. Kitt, *Schuetz-impfungen*.

BULL. AGR. EXPT. STATION OF NEB. VOL. II.

America. We know that it has occurred in Canada, and with all probability was introduced there by means of diseased swine from the United States. We also know that it is far more prevalent in what may be called the middle-western states than those north or south of the same, or those east or west of this section of the country. We know, further, that it has followed the extension of the railroads into the western states; but we do not know the limits of its dispersion in any single state, or even any county of any given state. This shows how much work we have on hand, not only with regard to the swine plague, but also all contagious and infectious animal diseases. In no single state in this Union is the annual loss from these diseases even approximately ascertained. Before this can be done, there must be vast changes made in the veterinary police laws of the country for the suppression and prevention of these diseases, and the selection of those persons intrusted with the execution of the same.

Such positions must be taken out of politics before any really valuable work can ever be expected.

Before such work can be done the nature and extent of these diseases must be actually ascertained by the inauguration of a trustworthy system of gathering statistics.

Nothing in this direction can reasonably be expected or demanded of the agricultural department at Washington until the respective states first inaugurate an individual system. Then, and only then, can correct general results be demanded of that department. The various "experiment stations" established under the "Hatch bill" should undertake this work, and the results form one of their annual bulletins under the law. It cannot be done, however, without a state law in each state supporting and making the work compulsory, and even state aid should be given to it. Nothing of this kind can be expected from the live stock commissions as at present formed, and under existing laws.

The history of the existing swine plagues of the world is a matter of very recent origin. The disease seems to have been at first recognized in this country somewhere in the forties of this century. The first investigations were made in England, in 1876, by Klein, and in this country by Detmers, Salmon, and Law, in 1878, but Detmers was the first to discover the micro-etiological organism. According to Klein, Pasteur began his researches about the same time that he did, but they had to

do with the "rouget," and not the swine plague proper, as will be shown later on. Although Loeffler showed the rouget to be caused by a bacillus, and not a "micrococcus," as Pasteur had claimed, it was not until Schütz positively demonstrated that another swine plague, and probably two, were caused by an ovoid-belted germ in Germany, that the swine plague proper was positively differentiated from the "rouget" (1885). Loeffler shares this honor with Schütz. Just how much relation these German investigations have to the true swine plague is still an open question, as will be shown in future considerations. That Loeffler investigated the "Wild-seuche," and that the first series of swine examined by Schütz were also complicated by that disease, is beyond all question. The clinical characteristics of the Wild-seuche most strikingly differentiate it from the true swine plague, as it occurs with equal freedom in cattle and deer, while the swine plague never attacks these animals. The "enormous oedema" of the Wild-seuche again differentiates it strongly from the swine plague, while we also have no satisfactory evidence that the peculiar caseous and destructive pneumonia seen in the swine plague, and especially the neoplastic, circumscribed tumefactions of the large intestines, have never been seen in the Wild-seuche. Up to 1878 these diseases were all looked upon as a form of anthrax. Bollinger first differentiated the Wild-seuche from anthrax by demonstrating the absence of the well-known "bacillus anthracis" in the tissues of animals dying from that disease. Professor Brown, of the Agricultural Department of the Privy Council of Great Britain (Report, 1886), writes as follows of the history of the disease. His remarks will be introduced without criticism:

"HISTORY OF SWINE FEVER.

"There is no evidence as to the date of the introduction of swine fever into this kingdom, but the fact of its prior existence in the American and European continents cannot be questioned, and it is probable that it was introduced here some years before it was detected in 1862.

"For a long period a fatal disease among swine in different parts of the world has been recognized and referred to by writers on diseases of animals. The descriptions which are given by the earlier authors are generally so vague, that it is not possible to identify the diseases which are referred to under the terms 'murrain,' 'leprosy,' 'anthracoid erysipelas,' 'anthrax,' or 'typhus'; but, in the light of recent inquiries, it is quite clear that the terms hog cholera, red sol-

dier, red disease, blue disease, purples, typhoid fever of swine, swine plague, and swine fever, are all names which are intended to refer to the same disease.

"America appears to have suffered to a more serious extent from this malady of swine than any other country. In a report which was issued from the Department of Agriculture at Washington in February, 1878, it was stated that returns of the existence of the disease (hog cholera) had been received from 1,125 out of the 2,440 states and territories in America; and the returns showed that, out of a total 18,987,342 swine in those counties, 2,599,542 suffered from 'hog cholera.' The money value of the losses from the disease, it was stated, was estimated at 10,091,483 dollars annually.

"Dr. Klein, in his report to the Local Government Board in 1878, enters on the subject of the history of swine fever at home and abroad, and he particularly notes that Spinola, in his work on 'Diseases of Swine,' published at Berlin in 1842, does not describe the disease.

"In 1857, Renault and Reynal's article, 'Charbon,' in 'Nouveau Dictionnaire,' etc., describes anthrax or splenic fever in the pig under the names red disease, gangrenous erysipelas, charbonous gastro-enteritis, typhus, etc., which are the titles sometimes given to swine fever even at the present time.

"In 1858, Dr. G. Sutton, of Indiana, wrote an account of swine pestilence in the North American 'Chirurgical Review.' In this article the ravages of the disease in the various states were referred to, and in evidence of the losses which were sustained it was stated that one distiller in Indiana had lost 1,400 hogs in one month.

"Hering, in 1858, describes a disease of swine under the name of 'malignant erysipelas,' which appears to be anthrax, and he quotes an account of typhus fever of swine from a paper by Falke. Some of the symptoms described appear to be similar to those which are seen in swine fever, but the account is imperfect.

"In 1865, Dr. William Budd, of Clifton, read a paper before the Royal Agricultural Society on the subject of 'Typhoid Fever of the Pig,' which was then prevalent in Somersetshire and also in Berkshire and Wiltshire. Dr. Budd's description of the symptoms and post-mortem appearances leaves no room for doubting that the disease was true swine fever. The illustrations in Dr. Budd's paper are remarkably faithful to nature.

"In 1869, Carsten-Harms, of Hanover, describes a 'red disease' of pigs, or 'swine plague,' which Dr. George Fleming considers to be identical with the pig disease which we are familiar with in this country. The writer was evidently, however, wrong in some of his conclusions; for example, in reference to the cause of the disease, which he maintains was due to the entrance of a fungus with the food. The

fungus, he says, may be detected in the epidermis of the diseased skin, in the kidney, in the bladder, and in the liver. Further, he did not look upon the affection as contagious; in fact, his experiments in inoculation were attended with negative results.

"In 1875, Haubner, of Dresden, describes a disease of swine (typhus) which resembled swine fever. Haubner agrees with Carsten-Harms that the disease is not contagious, and states that inoculation was followed by negative results.

"Professor Axe, in 1875, describes the disease, and considers that it is the exact counterpart of typhoid or enteric fever of man. In his experiments, inoculation with the products of the disease, or mixing them with the food, was always followed by its development.

"Roll, of Vienna, in 1876, follows Carsten-Harms, and states that it is not yet possible to decide whether the malady is to be put in the list of infectious diseases." pp. 4, 5.

The disease was first known to exist in France, as an idiopathic malady, in 1887, as shown by Cornil and Chantemesse, and later by Reitsch and his confrères. It appeared in Sweden and Denmark the same year, according to Selander and Bang. It has been reported in Russia, but I have mislaid the journal in which it was noticed. So far as is known to me, this comprises the countries in which swine plague is actually known to exist. According to the present literature, Germany might be considered still doubtful, but it seems to me that Roloff's observations should settle that question. This idea is strengthened by a remark in a letter just received from Doctor Hueppe, of Wiesbaden, in which he says:

"Recently we have observed a swine disease which appears to resemble the American disease." (In letzte zeit erscheint man, in F——, eine schweine seuche beobachtet zu haben welche der Amerikanischen näher steht. May 18, 1888.)

It will be thus seen that the first scientific knowledge we have of the existence of swine plague as an independent pest really dates from Detmers' discovery in 1878, as Klein has never described the germ correctly.

On the other hand there is some pretty strong evidence that that intellectual wonder of the ante-christian era, Aristotle, did somewhat differentiate between the existing swine plagues, upon purely practical grounds, and thus anticipated the work of scientific investigation by nearly 2,500 years.

The grand old Greek says :

"As to the diseases of quadrupeds, swine have three different diseases. One is known as 'bronchos,' and consists in an inflammation of the air tubes and masticating organs; it, however, frequently complicates other parts of the porcine organism, sometimes the feet become diseased and at others the ears. The disease develops rapidly, the swine soon losing their appetites. The herdsmen know of no other treatment than to cut out the diseased parts. Aside from this, two other diseases occur in swine. In one of them we may perceive pain and depression of the head, and in the other diarrhœa is the most frequent phenomenon. The last is said to be incurable." [That the swine plague is described in the above remarks is beyond all question. The other disease may have been either the rothlauf or Wild-seuche.] "In this disease, but few animals recover. Swine suffer most from 'bronchos' in the summer. Swine, also, have the measles. One can easily tell this, for they are to be found under the tongue."*

THE CAUSES OF SWINE PLAGUE.

The etiology of this porcine pest is not by any means the simple question to settle that it is with regard to the southern cattle plague or yellow fever. As the disease itself is a pathological complexity, when its secondary lesions are taken into proper consideration, so is its etiology a most complicated question. As of every other disease of animal life, so of the swine plague: it has many causes. Contagious and infectious diseases have, however, internal and supporting causes, aside from the one specific or exciting cause which is known as the *causa sufficiens*, without which no disease belonging to either of these great classes can come to pass.

These etiological moments are technically spoken of as—

1. *Internal Causes*, that is, specific peculiarities of certain species of animal life, which give to the individuals belonging to them an idiosyncratic susceptibility to certain diseases of a contagious or infectious character. These are congenital, or, as I prefer to call them, racial, causes. On the other hand, we have internal causes due to previous attacks of disease, or some weakness that was not inherent in the individual when born; the same are known as acquired internal causes. The latter have but little connection with the acquisition of the majority of contagious or infectious diseases.

2. *External Causes*, which includes every factor that may take

* Aubert and Weimer. "Aristotle's Thierkunde." Billings—Relations of Animal Diseases to the Public Health, p. 224.

part in extending a given disease among individuals or over an extent of country. This group is of vast importance in connection with contagious or infectious diseases.

3. *The Specific Cause*, which is a peculiar characteristic of diseases of a contagious or infectious character, and may now be positively asserted to belong to some form of micro-organismal life.

We have now to study the etiology of the swine plague in these three directions, and, therefore, will first give our attention to the so-called

INTERNAL CAUSES.

This side of the question of the etiology of any contagious or infectious disease is the one upon which we know the least, and shall probably always have to remain more or less in darkness about. It is one of those mysteries which, at present, at least, is beyond the power of scientific investigation to unravel. That it consists in some peculiar chemico-physical condition, which is idiosyncratic in each species, or peculiar to several, is beyond question. The demonstration of the *causa sufficiens* (great as such a discovery may be, and as useful as it may be made to humanity) in many diseases, instead of shedding any light upon these internal causes, has only rendered the dark clouds of our ignorance more overwhelming with regard to the same. While investigation has revealed to us the specific cause of anthrax, of glanders, of typhus, of Texas fever, of hen cholera, and the human cholera, as well as that of swine plague and other diseases, still we can no more answer the one great question, why horses are subject to glanders and cattle not; or, why the latter are destroyed by the rinderpest and the former not; or, why human beings have the scarlet, the mumps, the measles, etc., and the animals not, or why rabies is invariably a disease of the carnivora in its primary origin, and not in the herbivora; yet, as all these diseases have their known or unknown specific cause, we must assume that there is also some unknown condition, some racial, constitutional peculiarity, which offers the necessary conditions to the life of the specific cause in each of these species of animal life. This mystery, however, deepens when we consider a certain class of septicæmic diseases to which the swine plague belongs. When we come to discuss the specific cause of this porcine pest, we shall see, as has already been shown in our pre-

vious report upon the southern cattle plague, that the micro-etiological moment, the germ of the latter, like that of the swine plague, bear such close resemblance to one another that they cannot be differentiated either by microscopical study or in several of the artificial media in which they develop. But, again, we shall become still more perplexed when we find that in Germany there is a disease occurring among swine which is there known as the "Wild-seuche," in which the micro-organism, so far as I know, still more closely resembles that of the swine plague than that of the southern cattle plague. In fact, so near alike are the micro-etiological organisms of the cosmopolitan swine plague and the German "Wild-seuche" that almost all investigators have fallen into the error of looking upon them as identical complications, and hence have given Loeffler and Schütz the credit of having also discovered the microbe of the swine plague. With very grave doubts of my own course in this matter, I have followed in the same line in some anticipatory publications, not on account of the similarity in the micro-biological peculiarities of the organisms, for, as is known, I take a very positive stand against claiming identity upon that point alone, but because Schütz seemed himself strongly inclined to accept Roloff's description of lesions met with in swine, which exactly correspond to those called "characteristic" in this country and England, as belonging to his swine plague, and I have been inclined to think that Schütz only missed seeing them from the small number of swine examined by him, and his lack of extensive field observations. Since the earlier published sketches of my work, I have given this subject the most exact and critical study, and, as far as I can judge from reading the works of German authors, I am very strongly inclined to the opinion that Hueppe is correct when he claims that the Loeffler "Schweine-seuche" is the same as the long known "Wild-seuche," and hence that Loeffler has no claims for the discovery of the germ of the real swine plague. Unlike Hueppe, however, I do not base this opinion upon any characteristics or resemblances of the micro-organisms of the swine plague proper and the "Wild-seuche," but entirely upon clinical and necroscopical observations, the former especially. The so-called "Wild-seuche" attacks deer, cattle, and swine when exposed upon infected lands. The cosmopolitan swine plague never extends to cattle, no matter how severely exposed, under natural conditions. The "Wild-seuche" is

characterized by "enormous œdema," the true swine plague never is, although, as in most any case, some local œdema may occur at the *locus inoculationis* by artificial injection; it is never "enormous," however. The report of Schütz, as I shall show, is very uncertain in many points, and betrays quite a degree of groping in the dark, for several of his autopsies look very suspiciously like swine plague rather than "Wild-seuche," but of this later on. That my conclusions are probably correct, will find some support in investigations that have lately been going on in Denmark and Sweden, the published reports of which are as unsatisfactory as anyone could possibly desire. While upon this subject I will just mention the fact that we have, here in the West, still another cattle disease which is caused by a micro-organism belonging to this same ovoid-belted septicæmic group, which is even more malignant than anthrax, frequently killing the animals in a few hours, without anything but the slightest suspicious symptoms of illness. The matter is still more complicated in that swine have been among these cattle and remained well, so that it would seem as if the disease were not the "Wild-seuche," as I at first assumed, and there are good reasons for knowing that it is not the southern cattle plague. Of that disease at some other time.

Enough has been said, however, to show that in contagious or infectious diseases the most important internal cause is that peculiar physiological condition common to certain species of animal life, which must be of chemical nature, and which renders to the specific cause, of a given disease, the necessary nutrient materials to live and thrive upon.

Among the acquired factors, which though really not logically an internal cause, but still, under the circumstances, may be treated as such, are the large round worms which sometimes infest the intestines of swine, especially the small ones, *Echinorychus gigus* by name. While I have seen no reference to these parasites having gained any reputation among the swine breeders of Europe as the cause of swine plague, quite the contrary is the case here, especially in the western states, where a large proportion of the farmers are still more or less of the opinion that these worms are the real cause of swine plague, and the country is overrun with peripatetic "hog doctors," who make quite a living peddling their so-called "hog cholera cures," and preaching their verminous doctrine to the farmers. When these

gentry can find a hog that has died of the swine plague, and on opening such be fortunate enough to find the small intestines filled with *echinorynchi*, their fortune is made in that district; the farmer cannot refuse to believe what he sees with his eyes, and consequently buys a lot of worm powders, and, should it happen that the outbreak among his swine had pretty well run out, or be a mild one and soon terminate, he is sure to give the credit to the "worm doctor," and until the charm is broken by bitter experiences in the same region the praises of his wonder-cure are on every farmer's tongue.

The only question of interest in connection with these parasites is, do they play any part, through wounding the intestine, in causing the swine plague? To my mind this question is to be positively answered in the negative:

1st, Because they do not cause any serious wounding of the intestinal wall. They seem to fix their proboscis in the opening of some follicle, where they attach themselves and cause more or less chronic irritation of the tissues around, which is sometimes accompanied by hemorrhage, but the mucosa remains intact, and I have yet to see any evidence of superficial traumatic lesions of the mucosa which would warrant the hypothesis that the bacteria of swine plague gained access to the circulation in this way.

2d, The lesions, of an ulcerative or neoplastic character, are entirely limited to the large intestine, except in the most acute cases; when they are of a most extended diphtheritic character, no part of the intestines entirely escaping.

Again, the presence of these worms in the intestines of swine is not nearly so frequent as these "worm doctors" have made the farmers believe. In one hundred autopsies made upon diseased swine, of which a record was kept for these worms, I found them but twice, though sometimes I have met with quite a number in succession in other cases since then, especially among pigs under three months old, but it so happens that most of my necroscopical examinations have been made upon older animals.

Dr. Detmers, who has had a very extended experience, says:

"The absence of worms in 75 per cent of the whole number of animals examined would seem to prove conclusively that the morbid changes in the swine plague cannot be attributed to the work of entozoa." U. S. Ag. Report, 1870, p. 365.

Whether the existence of traumatic lesions in the mouth and fauces has any etiological importance, is a matter I do not feel competent to decide at present, but like trauma in the skin, this way of infection would seem to be of minor importance, if we may judge from the many negative, or partially negative, results which often follow the subcutaneous introduction of small quantities of pure cultivations of the bacteria, unless the same be unusually malignant; for this reason I have invariably resorted to intra-abdominal injection in all but a few of my earliest experiments, especially as it seemed probable that in this way intestinal lesions would be the most likely to result and thoracic less certain, but in this regard the results were as severe in the lungs as elsewhere, if the animals survived long enough.

We may now consider that most important of all the causes of the swine plague:

THE EXTERNAL.

Without entering upon the discussion of the question of the "contagiousness" of swine plague at all in this place, I will simply say, that I positively and unequivocally deny any contagious character to the disease, and look upon it as an extra-organismal septicæmia; that is, a disease which finds its specific origin entirely and unequivocally outside of the porcine organism, or, in other words, the specific cause finds its primary locus of development in the earth and earthy materials, and never within the hog, until it has entered and infected it from these outside media.

The external causes of swine plague, then, are everything that can possibly exert any influence in extending the disease over a section of country by dispersing its specific cause, or anything which can possibly protect or support the life of that cause, the specific germs of swine plague, which are of themselves an external cause.

Among these factors or causes may be mentioned, as first in importance, the diseased swine; then the pens where such are or have been; material from such pens conveyed to other places which offer favorable conditions for the preservation of the life of the specific cause or its further development; anything which can convey such material away, visitors or owners who go to visit their neighbor's hog yards after being in infected pens, dogs, fowls, rats, crows, or hens; and lastly, the conveyances of common carriers, cars, wagons, etc., that have been used to convey diseased swine, or manure, or

straw from infected pens; the cadavers of sick swine, and water-courses running through or by infected hog yards. All these factors have been known to, or are said to, have played a part in causing the extension of swine plague over this country, and therefore deserve our attention. It is self-evident that if we could absolutely prevent all these causes coming into action, that the prophylaxis of swine plague would be an easily settled question, without the necessity of our knowing much of anything about the specific cause, but these factors have been more or less thoroughly known for many years, and yet nothing effective has ever resulted looking toward the better protection of the hog raiser. It is partially his own indolence, but more largely that of our governing bodies, which have taken no proper steps for the regulative prevention of this porcine pest. Still, the example and experiences of European countries with regard to the rinderpest of cattle, and the black death and bubo pest of our own species, which have been driven out of Europe by the proper appreciation of these supporting, or external, causes, shows what can be done to protect a people from the ravages of disease by a true and observing government, even though nothing is yet known of the specific cause of these diseases and quite a number of others that have been restricted with nearly the same success, by a proper regulative treatment.

DISEASED SWINE AS A CAUSE OF SWINE PLAGUE.

Although I shall have to take up this question in detail in the consideration of the nature of this porcine pest, there is another side of the question which should be treated upon here. First, however, let me say why the swine plague is not a contagious disease. Because it does not find its primary origin in diseased swine, and extend directly from hog to hog. Although it does not do this, still diseased swine are the first and primary factor in the extension of the disease. Because of this fact, inexperienced and incompetent observers have been utterly unable to see that sick swine do not infect healthy ones by direct contact. We, who have experimented, know that infection by the subcutaneous inoculation of healthy swine is very uncertain indeed, so that this manner of transmission from diseased to healthy swine is absolutely excluded. Then, there is only one other method left, and that is that healthy swine shall aspire the breath of those

diseased. Every swine breeder in the West knows that a fence will keep the swine plague from extending from diseased to healthy swine in that way. Practical evidence will be introduced to illustrate that fact. Then how do the diseased swine act? Were the disease contagious, it would extend from swine to swine in some way, no matter how carefully we carried out the disinfection of their surroundings. In my experiments I have fully tested this question by putting diseased and healthy swine so near together that nothing but a wooden pen separated them, the diseased being surrounded by healthy hogs, but nothing within the pens of the former but the air could get out, and yet not a single case of swine plague has resulted, except where I wanted it, within the pens. The fact of the matter is this, healthy hogs become infected from the land, or earthy refuse of places where diseased hogs have been; they then, through their manure, in the first place, and urine, in the second, add new fuel to the fire, the germs multiplying in their intestines again being planted on or in the earth, and wherever such swine go they do this thing, just as southern cattle do in the southern cattle plague. It matters not how we introduce the disease germ into swine, the constant examination of the faeces shows that the germs get there when the animal becomes diseased, just as I have shown to be the case in the southern cattle plague.

EARTH, PENS, AND INFECTED REFUSE THE CAUSE OF SWINE PLAGUE.

The assertion that the swine plague is an *extra-organismal infectious septicæmia* has called out the following denial from the erudite bureaucrat who rules over the fate of our great live stock interests at Washington:

"The same author insists in the most positive terms that hog cholera is an extra-organismal infectious disease. In other words, that the parasite is one the natural habitat of which is the soil, that the hog obtains it from the soil and not through contagion, and that once planted in the soil this microbe remains there and multiplies for an indefinite period. Where are the records of the experiments which demonstrate this proposition put forth in such emphatic terms? Have you seen them? I have not.

"In the laboratory of the bureau of animal industry we have plodded along for three years laboriously endeavoring by means of experiments to throw some light upon the biological characters of this microbe and the conditions under which it may be preserved. We

have observed after many outbreaks that fields and pens have been safely used within three to six months after the disappearance of the disease." [Tell that to a Western farmer and see how long you would live!]*—American Veterinary Review, April, '88, p. 15.*

"The same author" being myself, I will endeavor to throw some light into the clouded recesses of Mr. Salmon's mind, for it is certainly very evident that, after investigating the swine plague since 1878, the worthy gentleman has only been successful in contradicting himself and mystifying the all-suffering public more and more by his very erratic annual announcements.

Mr. Salmon wants proof! He need not have cried so boldly for experimental evidence, for had he had any observational powers, whatever, had he, in fact, spent one honest three months out of these ten wasted years, in the study of swine plague as it exists under natural conditions, he would have found that the hog raisers of this country have piled up mountains of experimental proof which shows that swine plague is an extra-organismal septicemia and nothing else. Had he even studied his own records of former years, he must have come to the same conclusion. It is one of the most strikingly singular phenomena that, when one wants evidence against any of Mr. Salmon's assertions, that the best place to seek it is among his own writings, and as I am very averse to being partial in the selection of testimony, we will, as Priscilla asked of John Alden, request the gentleman to speak for himself. Under such favorable conditions he will tell us the following instructive and interesting facts:

1. "It seems probable that the contagion may be preserved over winter in manure, straw, litter, or in the remains of unburied animals which have died during the preceding year." *U. S. Ag. Report, 1878, p. 440.*
2. "The regulations (preventive) should go into effect in winter or early spring, when the fewest animals are affected, or when, as my experience indicates, the disease is entirely extinct." *Ibid., p. 442.*
3. "In what direction shall we look for this much to be desired remedy? Can we disinfect the great hog pastures of the South and West?" *Ibid., 1883, p. 56.*

Having put Mr. Salmon on the witness stand, it is now incumbent upon us to ask that gentleman to explain himself.

1. Does Mr. Salmon know of any contagious disease of animal life, the germs of which will retain their active or contagious char-

acter in full vigor when contaminating such loose material as "manure, straw, or litter," and preserve this quality over winter?

2. Does he know of one in which the germs will retain these active qualities in "unburied remains which have died during the preceding year"?

3. Did he ever hear of a single contagious disease of animal life which ceases to pass from animal to animal "in winter or early spring, when, as my (his) experience indicates, the disease is entirely extinct"?

4. If a contagious disease, why does he speak at all of disinfecting "the great hog pastures of the South and West"?

To the readers it should be said, does not Mr. Salmon, in these very quotations, admit that the germ finds its development outside of the animal organisms? Does it not find the favorable conditions to its life in the manure, the hay, the straw, the litter, and the soil of the "great hog pastures of the South and West"?

It is a well-known fact that the germ of any strictly contagious disease will soon lose its vitality under just these circumstances, while those of anthrax and all such diseases, which find their primary place of development in the soil or earthy material, will live much longer outside of the animal organism, and retain their disease-producing qualities, than those of any contagious disease, which are constitutionalized to an unvarying kind of nutrition and a constant degree of temperature.

It is a well-established fact that the thermal limits under which the germs of contagious disease can be developed, extra-organismally, are very narrow, being but little under that, or over that, but especially under, of the animal body, while those of the strictly infectious diseases, like swine plague, permit quite an extreme degree of latitude in this direction. Most of them will develop, in suitable media, anywhere between 20° and 40° C., and these are by no means the extreme limits, while those of active development in the germs of contagious diseases may be said to be between 35° and 41° C., though some slight variation may occur in either direction. To illustrate this point it is not necessary for me to give the exact limits.

The period or locality of the greatest extension of contagious diseases among susceptible species of animal life is just when or under such circumstances as Mr. Salmon tells us that, "in his experience," swine plague is entirely extinct, that is, in the "winter," or colder

months, when the animals are either housed or collect closely together in order to keep warm. It is the congregation of animal life in close apartments, or the opportunity to close contact, which causes the rapid extension of small-pox, measles, mumps, scarlet, spotted typhus, and, last of all, syphilis, among the human family, and it is the same conditions which lead to the rapid extension of glanders, pleuropneumonia, the rinderpest, etc., among those animals susceptible to them. The season of the year has nothing to do with it, except that the summer months are less favorable to their extension, because the animals are not, generally, so closely housed, and hence the chances of mediate or immediate contact are less. Some unreflecting state veterinarians assume glanders has diminished every spring, when the complaints become less, only to find out their mistake the succeeding winter. The degree of contact necessary is dependent upon the manner of action of the contagious principle. Now, under those circumstances in which contagious diseases acquire their greatest extension, we see that Mr. Salmon admits, though incorrectly, that the swine plague becomes "extinct," and it is a well-known fact that, though outbreaks may and do occur in those months, still that the disease does not, or is not extended at such times from one place to another, except under very exceptional circumstances. I know of several instances where the healthy hogs have been taken to places where such material was, and not a hog of any kind had been there for from one to three years, and yet swine plague resulted.

Now Mr. Salmon wants proof that it is the "land," the manure, the litter, and not per contact with diseased animals, by which swine plague is communicated. He says, "Where are the records of the experiments which demonstrate this proposition?" "Have you seen them?" he boldly asks an audience that knew even less about swine plague, and still less about pathology and micro-biology, than he does. Then he tells his audience, "I have not." Mr. Salmon's answer sufficiently shows not only how well he has studied the swine plague, but how honest he is towards the work of other observers, the very names of whom he takes care not to mention, because he is afraid some one might have curiosity enough to read what had been said. As I happen to be "another authority," or "the same author" to whom Mr. Salmon alluded so kindly, permit me to say that proof, having the value of the most exact experimentation, has been given in my writings

anticipatory of this report. Proof so strong that no experimental evidence could be more conclusive. The farmer always values "practical experience" far beyond the results of "those scientific fellows," and I have the same weakness when I find the same on all sides.

PROOF THAT SWINE PLAGUE IS AN EXTRA-ORGANISMAL INFECTION, AND NOT A CONTAGION.

In my necroscopical notes is mentioned an outbreak of swine plague at Valparaiso, Neb., in December, 1886, the history of which is as follows, but first let me say something about the weather that prevailed then and before that time: On November 17, 1886, we had the most severe snow storm I have yet seen in Nebraska; the weather was very cold, and the snow deep and drifted very high. This was followed by a thaw, which took off some of the snow, when the ground became bare, and froze up hard as stone, and remained so until into January. Before and after December 1st this was the condition, the thermometer being in the vicinity of 0 F. all the time. Under such conditions, according to Mr. Salmon's "experience," swine plague should have become "entirely extinct." Now let us see if the facts will support any of his "experience." In the fall of 1885 there were about 200 hogs in a pasture of about an acre, in the town of Valparaiso. Upon this pasture was an old barn; the hogs ran over the ground and slept and lay in the barn, the ground floor of which was covered with straw and litter. This lot of hogs became diseased with the swine plague, and a very large number died. As is well known, sick hogs seek the protection of straw and litter, and, as the weather certainly was not warm, they collected in the old stable. Nearly all died, and were carted off; the balance were sold; so that on the 1st of December, 1885, there was not one left on this piece of property. The land was so situated that no other hogs were kept in the vicinity. From December, 1885, to December, 1886, not a hog of any kind was on that land or in that barn!!

Seeing the place vacant, a Mr. J., who is the man of the place, running a general store, grain elevator, etc., and who owned and leased quite a number of farms on shares, having thus a big amount of corn on his hands, thought the best thing he could do was to lease this piece of land for the winter and put his corn into pork. The place was most favorably situated for his purposes, being near his store,

and his clerks could throw the corn in to the hogs without causing any extra expense for labor. Mr. J. knew all about the disease having been on the place the year before, but he had probably read the reports of the U. S. Agricultural Department, and like many of his neighbors thought these "reports" could be relied on, the general idea having become extended over the country by this means, that swine plague "is a contagious disease," or, as these pathological wonders call it, "a contagious fever," so, like many another who has paid dearly for his music, he reasoned: as there has not been a hog on that place for a year, there can be no danger; with this idea in his mind, Mr. J. went out and bought about 200 shoats under four months old, and knowing every acre of the country round about, and being the proprietor of the general store of the town, he also knew all about the troubles of every farmer, so he was extremely careful in purchasing these "shoats," and did not buy one of any farmer that ever had the swine plague on his place. The "shoats" came in and were delivered in wagon loads. The weather being bitter cold, they immediately made a rush for cover in the old barn, and, as it was told me, "kicked up a fearful dust" in this old dry straw and litter. Suffice it to say, that within ten days they began to sicken, and nearly all died of swine plague in a very malignant form. It was further known that no swine plague occurred subsequently upon the farms where they were purchased. Now this old litter, over a ground floor, offered just the conditions necessary to keep the germs protected from the severe cold existing, and from too much dessication in the summer. I will only say that the ovoid-belted organism was found in this case, which also is a striking "aspiration experiment," one of the most marked lesions being acute pneumonia of a bronchial type.

Again, I desire to call attention to one fact, upon which Mr. Salmon and myself agree, and that is, the uncertainty of subcutaneous inoculations of pure cultures upon healthy swine. Now when the material is taken from malignant and undoubted cases of the disease, and as swine are the animals in which the disease occurs, it is evident to every competent experimental pathologist that such uncertainty in results could not exist in a strictly contagious disease. This "uncertainty" can be easily avoided by the injection of even smaller quantities of pure cultures directly into the abdominal cavity, as I have nearly always done, or into the thoracic organs; Cornil and Chantemesse.

Another case !

Some seven miles from Lincoln, Neb., is a farm which was bought by a Mr. A. some four years since, but who put a heavy mortgage upon it, as is the way the farmers try to get wealthy here in the West. He at once built hog-pens and borrowed still more money to buy hogs. Among them he bought the swine plague, and it swamped him, and he was sold out by his creditors. For three years this farm remained unoccupied, and the nearest neighbor was over a mile away. The creditor who owned the farm let it to a man last fall (1887) rent free for one year. The farmer bought hogs and put them into the previously infected pens, they became affected, and nearly all died. Does that look like contagion? The man bought assuredly healthy hogs, as I know where they came from, and the places are still free from swine plague—May, 1888.

Now for another case, that every one about me knows all about, and which I had under constant observation for the past two years.

December, 1887, swine plague broke out at the "College Farm," where my experimental station is, but among the thoroughbred hogs, in their own pen, and not among mine, which were kept at a distance and had their own attendant. Not a case of natural infection has ever occurred among my experimental hogs since I have been at work, and the last case of intentional disease occurred in August, 1887. Previous to, during, and since this outbreak among these thoroughbred hogs, my own have been perfectly well, so that excludes every possibility of infection coming from that source, as there was and is quite a stock of young animals among them awaiting future use.

Mr. Wing, superintendent of the farm, writes me the following history, under date of February 18, 1888 :

"In the fall of 1884, November or December, occurred the first outbreak. It raged with more or less violence until about February 1, 1885, at which time all but ten hogs on the place had died. The hogs were at first in the hog house and pens adjoining, but were removed to the cattle yards, some dying in both places. In the spring they were all moved back to the hog house. In April, 1885, a thoroughbred sow was bought and placed in the pens where the hogs had died during the winter. She died May 4, 1885. In December, 1887, some pigs born in August, descendants of the survivors of the outbreak of 1884-5, commenced to die. They were in the hog house and pens previously mentioned, and with their progenitors had been kept there constantly since the former outbreak. Twenty-two died."

The character of this outbreak will be noticed again in my necroscopical notes and remarks on prevention. As no new stock had been introduced among these hogs, and as my experimental stock was kept in the cattle yards and were and are healthy, and as there was no disease among the thoroughbred stock from May 4, 1885, to December, 1887, and as they were constantly under the watchful eye of Mr. Wing, all possibility of contagion is absolutely excluded, and the only way or locality from which they could possibly have been affected was from the infectious principle in the pens from the previous outbreak of 1885. Why the disease did not appear in 1886 and 1887 is something we cannot answer. But the very fact that the germs will lie apparently dormant or inactive, for a year or so, and then cause such an eruption as this, is the strongest possible proof of my conclusion that swine plague is not a contagious disease, but, on the contrary, an extra-organismal disease. Just such unaccountable phenomenon occur in anthrax, typhus, cholera, and other diseases, which belong to this class, though caused by a different species of organism, morphologically speaking. We do not yet know enough to correctly appreciate the influences of heat and cold, moisture and dryness upon the eruption of those diseases, the specific causes of which find their place of primary development in the earth or earthy materials. Although it will be alluded to again in another place, I wish here to call attention to another practical experience, which finds ample support at the hands of many intelligent swine breeders of this state, and that is this, that so long as they treated their hogs according to teachings of Mr. Salmon, that is, as if it were a contagious disease, and removed the sick ones from among the apparently well, they never checked the outbreaks an iota, but when with equal care they followed my instructions, and took the well ones out of the infected pens, and left the sick ones there, the further extension of the disease among the former was stopped at once. In many cases not a single one has been lost among those taken out by men of experience and exact observation. Now, were swine plague a contagion, such successful separation would be an impossibility.

This fact can be so repletely supported by testimony of practical hog men, that it needs no further notice, and it would be superfluous to introduce it here.

Before introducing further practical observations on my part, it

may be well to quote some more evidence from Mr. Salmon. In his article on "How to prevent hog cholera," he says:

"There is consequently very little about a pen in which the virus, when scattered in the discharges of infected animals, will not increase in quantity, and form a potent source of infection." Report 1886, p. 654.

"In the first place there should be no communication between infected herds and such as are still free from disease. The virus may be carried in various ways, even on the shoes of persons. A small quantity thus introduced may multiply in the soil and water until it becomes a center of infection for many animals. Streams into which sick animals have dropped their discharges, or in which dead ones have lain, must be considered as vehicles of the disease for all herds below the source of infection." *Ibid.*, p. 656.

"When the disease has appeared in a herd, the ground upon which the animals have lived at the time must be considered as infected, and it is much safer to remove all the well ones to uninfected grounds than to simply remove the sick." *Ibid.*

"The blood and fluids from carcasses would be certain death; that the blood and fluids from these dead bodies contain the virus, and when scattered over the soil, or thrown into streams, they simply distribute the virus, allow it to multiply, and all other animals are thereby put in the way of becoming infected." *Ibid.*, pp. 656-7.

"As it is quite impossible to disinfect the soil with any degree of certainty, it is very desirable that the still healthy animals be transferred to fresh ground. In this way the dangers arising from an infected soil are averted." *Ibid.*, p. 658.

Read the next!

"We do not know that the virus can live in the soil through the winter, but it is highly probable. Hence, thorough disinfection will practically lessen the chances for a re-appearance of the disease in another year." *Ibid.*

In another place Mr. Salmon has told us that such disinfection is quite impossible! Can any further evidence be desired? Here are Mr. Salmon's true ideas in optima forma! We quite agree with them! It is "soil!" "soil!" "soil!" and scarcely a word about sick hogs! No mortal human ever heard of using such language with reference to a contagious disease! There it is sick animals! animals! animals! No one ever heard of a contagious disease infecting the soil! I use the word "infection" in the sense to multiply in, and to keep multiplying in, not merely to drop on and stay there a little while.

The fact of the matter is, Mr. Salmon was simply trying to make a point before an incompetent and unreflecting audience when he challenged my statement that swine plague is an extra-organismal-infectious septicæmia. There is not a bit of evidence in his writings but what confirms that idea, and none that the disease is contagious.

MORE EVIDENCE THAT THE LAND AND REFUSE ARE AMONG THE
CAUSES OF SWINE PLAGUE.

In the summer of 1886 I visited an outbreak of swine plague at the town of Crete, Neb. The animals belonged to a Mr. N., one of the most intelligent and observing farmers I have ever met. I found the hogs were upon high land. The owner had lost many in the same pens the previous year. On adjoining land, separated only by a fence, but so situated that the drainage was all toward the infected yards and pens, Mr. N. had a large number of thoroughbred breeding swine; the fence was not so tight that the animals could not smell of one another. I remarked to Mr. N. that he should be more cautious about these fine animals, that some people said "the disease was contagious." He immediately answered "There is no danger, a fence will keep it off from them if we are careful not to go in among them ourselves."

Fences do not keep off contagious diseases, and this evidence of a man who had had thousands of dollars worth of practical experience has all the value of the most exact scientific experimentation, especially when supported by hundreds of similar experiences from other men.

Mr. N.'s remark, "That owners must be careful not go into pens or fields where healthy hogs are, if they have diseased ones in another place and have been among them," brings to mind another piece of empirical evidence, which has all the value of the scientific proof which Mr. Salmon is so desirous of seeing, and which has been published in some of my previous papers.

Mr. B., living four miles south-east of Lincoln, had never had the swine plague on his farm. His brother, who lived some four miles away, had the disease very severely on his farm in the fall of 1886. On a rainy Sunday Mr. B. took his team and drove over to his brother's, to see how his hogs were getting on. The two men drove into the hog lot, and got out and looked over the affected herd.

Everybody should know what Nebraska mud is. It sticks to one's boots and horse's feet and wagon wheels like so much thick paste. After their inspection, these two wise men got into the wagon and drove over to B's. They drove in among his hogs, got out and looked over them, and unhitched the horses in the hog yard, leaving the wagon there. In about three weeks B's hogs commenced to die of swine plague. There was no swine plague nearer than his brother's farm. There was no means of communicating the disease to B's hogs but the dirt brought from his brother's farm in the way indicated.

A practical swine breeder says:

"I heartily endorse D. L. Thomas' statement as to hog cholera not being contagious. I do not believe it is. I had six nice thrifty pigs about five or six months old, in clean place to sleep and eat, and a yard of about 30x100 to run in. My neighbor on the south had two pigs about eight or nine months old, in a dirty, filthy pen, and they had the cholera. My pigs could go right up to the pen, which was on the line. My neighbor on the north had quite a number of pigs, and lost ten or twelve with cholera, and my pigs never had anything wrong with them." W. K., in *Breeder's Gazette*, Feb. 29, 1888.

Is this experimental evidence enough, Mr. Salmon?

THE INFLUENCES OF SEASONS, HEAT, AND COLD, AND RAINS, AND MOIST SOILS UPON SWINE PLAGUE.

In my earliest investigations of this disease, and before I knew much of anything about it, I was struck with the fact that all the earliest outbreaks, in the spring of 1886, in the vicinity of Lincoln, were occurring upon the high land, especially upon the crests of the uplands, and as the warm weather approached it was observed that it gradually appeared on farms situated in the valleys or on level land. This so exactly corresponded with experiences in anthrax, that it at once caused me to conclude that I had to do with a land-infectious, instead of a "contagious" disease, as I had been led to think by reading the reports of Mr. Salmon, and so I followed the matter with especial interest. This spring was excessively wet, as well as is the present one, when the same thing is being repeated. Cold, wet lands are not favorable to the early eruption of the swine plague, any more than they are to anthrax. It is only when the ground water has sunk through drainage and evaporation (these lands all had a southern exposure, and hence became warm and dry early in the season) that

the disease again appears. In every case the disease had been upon the same land in previous years. It is only when the weather is very warm that moist lands are favorable to the development of germ life, and at such times high and dry and exposed lands become less favorable.

While I have been unable to get any reliable data upon the influences of rains upon the eruption or extension of swine plague, other than the above, that reliable investigator, Dr. Detmers, seems to have been more fortunate. Upon this subject he says:

"Arriving at Champaign, Illinois, I found that the disease, which had never entirely ceased to exist in the county since July, 1878, was spreading very slowly, but had made a temporary stop, or ceased to spread, immediately after each heavy or pouring rain. I found further, that its propagation within the herd become visibly slower, or stopped altogether for several days after each violent rain storm in all such herds as were kept in a pasture or hog lot where the water could flow off, but that the spreading was not visibly interrupted in such herds as were kept in a timber lot or in a pen under roof." U. S. Ag. Report, 1879, p. 382.

"The losses during the winter and until spring in Mr. M.'s herd were not very severe (it had been there the previous fall), but, in the spring, after the sows had farrowed, Mr. M. lost most of his young pigs from the disease, but only a few of the old ones, which is not strange, if we take into consideration that the season—a cold winter—had not been favorable to the propagation of the infectious principle.

"As soon, however, as the heavy spring rains set in, the disease ceased to make much progress—at any rate from May till August but few cases occurred. Ibid., p. 384.

"One thing is certain, immediately after a heavy or pouring rain, a perceptible stop, or cessation, could be observed in the spreading of the disease, while each time after the lapse of about a week a renewed spreading took place, to be interrupted only by the next heavy or pouring rain." p. 392.

"Swine plague, until the last of December, or until the ground became covered with snow and the weather exceedingly cold, was spreading from farm to farm and from place to place, but as soon as the temperature commenced to remain below the freezing point, at noon as well as at night, it at once ceased to spread from one farm or locality to another. At the same time, however, it was also observed that the very cold weather of the last days of December and the first of January did not materially interfere with the spreading of swine plague among the animals in all pens and hog lots in which the disease had previously made its appearance, and in which the way of

feeding and watering the animals was such as to allow contamination of the food and water with the excrements or other excretions of the diseased hogs, or in which the hogs and pigs still healthy had open wounds or sores, and had to sleep together with diseased hogs, in the same sleeping place and on the same litters. Afterwards, when milder weather had set in, the spreading from one place to another very slowly commenced again." p. 381.

"Mr. W. had swine plague in his herd a year ago last winter (1878), and disposed of every hog and pig he could find on the place. He commenced anew, and bought twenty head of healthy young pigs. After receiving them, one dead pig, belonging to his old herd, was found stiff and frozen in a fence corner, where it had died. It was immediately buried three feet deep, but in frozen ground, and there the carcass remained frozen till the latter part of winter, when it was found unburied and consumed by the twenty healthy pigs. Ten days later they commenced to die of swine plague." Detmers, 1879, p. 395.

I inoculated agar agar tubes from the spleen of two hogs that had died from swine plague, and buried three days in the snow of the great snow storm of November 17, 1886, near Lincoln, Neb. The carcasses were frozen hard, and the spleen was so hard as to break in two on being bent. The characteristic bacteria grew plentifully in forty-eight hours, and were fatal to mice in twenty-four to thirty-six hours.

Since then the same experiments have been repeated over and over again. Cultures have been frozen solid for forty-eight to ninety-six hours without it having any effect upon the virulent activity of these micro-organisms.

Professor Law's experiments show the same results. U. S. Ag. Report, 1878.

THE INFLUENCE OF RUNNING STREAMS UPON THE EXTENSION OF SWINE PLAGUE.

That swine plague has been extended in this way has been and is frequently asserted by many practical farmers, as is shown by the following examples:

Mr. J. C. D., of Lincoln, writes:

"I have to say I am the only farmer that lost no hogs and had none sick in 1885-6—fully two-thirds of the hogs in this vicinity having died. My neighbors turned their sick hogs out, and they were frequently around my pens, and twice broke into them, but I never lost one. I don't believe in medicine. Stevens creek runs through my farm, but I never let my hogs near it, but water them

from a well. The plague appears on the creek first and spreads rapidly down it, appearing later on the highlands. Turning diseased hogs loose to die in the draws, to be washed away, and dogs carrying the carrion about, contributes largely to spread the disease."

Contagious diseases are not spread by streams, but by diseased animals directly. Detmers gives the following illustrative examples:

"Mr. Henry Yothy informed me that his next neighbor lost every hog but one on his place; while he (Yothy) had nineteen head of swine up in a yard, and did not lose a single one, notwithstanding his neighbor's diseased animals had been running at large and trampled all around Yothy's pens and close to his fence every day, but as Yothy's hogs had no lesions or wounds whatever and had remained separated from them by a fence, and had no opportunity to consume food or water soiled with excrements or urine of the latter, or to become infected in any way."

The next examples are especially suggestive as to some necessary preventive measures.

"Mr. F. has no near neighbors. Several streams have their origin on his farm, but only one has its source above and runs through it. He has always remained free from swine plague until last year, 1878. Three-quarters of a mile from his place, situated at the head of a ravine, which, however, does not extend through Mr. F.'s farm, is a rendering establishment where dead hogs are rendered into grease and lard oil. The carcasses of dead hogs are cut up there, and pieces are frequently lying about on the ground, and those parts which do not contain any grease, or are worthless, such as the lungs and intestines, are thrown into the ravine and are washed away by the water or remain where they are thrown until it rains. Further down this ravine unites with another, and these two make a small creek which empties into the Mississippi. Every herd of swine that had access to that creek became affected, and nearly every animal died."

Mr. H. makes the following statement:

"I have a farm on the banks of the Henderson river, and last year kept quite a herd of hogs. One morning I found, lodged at my hog lot, which joins the river, a dead hog which had come down stream. My hogs discovered it earlier than I and were feeding on the carcass when I came. Ten days later they commenced to die. Loss, \$1,500."

THE INFLUENCE OF STOCK CARS IN EXTENDING SWINE PLAGUE.

That cars which have been used to convey diseased swine have been the cause of extending the disease into previously non-infected dis-

tricts of our western states is a fact too plain to be questioned. In fact, the extension of swine plague into such distriets has been directly in correspondence to the extension of the railroads, and from them it has gradually extended further into the country.

A Mr. V. informs me that the first cases of swine plague in the Ozark mountain country, in Missouri, was due to some freight cars that brought railroad iron there to a road that was building. The bottoms of the cars were covered with straw and litter, which was pulled out in unloading the rails. The disease first appeared in the hogs of railroad hands living in shanties along the line of the road, and gradually extended to those of the settlers more distant therefrom. Similar ooccurrences have been reported to me from the western part of Nebraska.

RATS AS A CAUSE OF EXTENDING SWINE PLAGUE.

In this regard I have a most striking example:

A Mr. ———, a friend of mine, is an extensive breeder of fine swine. Being short of corn, he bought a lot of old, rat-eaten corn from a neighbor that had lost his swine from the plague. Soon after beginning to feed this corn, his swine commenced to sicken and die. Removal of the well ones and change of feed saved the balance. It is a little singular that only those to whom the owner gave fermented corn should have become infected,—at least so the story is given. However, on examining this corn, it was found full of rat manure, and from this manure I obtained the germ of swine plague, and by bouillon cultivation and then inoculations of mice got it in a pure condition, and then produced positive results in a three-months-old pig by intra-abdominal inoculation, getting the same organism again from this animal's blood and organs.

In this connection it may be said, also, that dogs and fowls have been accused of carrying the inficiens, or infecting material, from one place to another, but I have no positive evidence to give in that direction.

Having thus called brief attention to the supporting and extending causes of swine plague, the next question of interest is, naturally, the specific cause, the "*causa sufficiens*," without the existence of which all the etiological points, or factors, previously considered would have no value whatever.

THE SPECIFIC CAUSE OF SWINE PLAGUE.

The historical review of the investigations, into the cause and nature of swine plague, that have been conducted in various parts of the world, is a matter of the deepest interest, but in no country of greater importance than the United States. Nowhere else have they been characterized by such manifest inefficiency, and in no other country, or among any other class of investigations, upon any contagious or any infectious disease of animal or human life, have these investigations been of a fraudulent and deceptive character. With the exceptions of the researches which I have had the honor to conduct, under the auspices of the state of Nebraska, and under the direction of the Regents of the University of that state, the investigations into the etiology and nature of the swine plague in this country have been conducted under the control of the United States Department of Agriculture, the workers engaged by that department having been doctors Detmers and Law, in the past, and the present chief of the bureau of animal industry, Mr. D. E. Salmon. They began in 1878, and are still going on. It is my purpose to consider the work of Mr. Salmon as a whole, particularly as it has had the effect of crowding the reliable work of Detmers entirely out of sight, and been mistakenly taken as correct by the majority of the patho-bacteriologists of the world, having had the weighty endorsement natural to the publication of such work by a department of a government such as that of the United States. As said, Mr. Salmon began his work for the Agricultural Department at Washington in the year 1879, the first announcement being in the report of that year, in which he has nothing to say about its etiology, but considerable as to its character, which is especially interesting from its bearing upon the latest conclusions of that person in the report of 1886 (issued in 1887). Although it will be necessary to consider this part of the subject in detail in another place, it is proper here to call attention to the fact that in his last report, and subsequently, Mr. Salmon vainly endeavors to show that we have two porcine pests in this country, one of which he now calls "hog cholera," and the other "swine plague," while previous to this time he had never known of but one "swine plague," and combated the term "hog cholera" as a misnomer, notwithstanding the fact that he had been busy studying the disease almost continuously since 1878. His

reasons for this sudden change of opinion will be shown later on. The fact that European investigators have accepted Mr. Salmon's erroneous assertions as authoritative, makes it my imperative duty to my country and confrères in this branch of investigation to place the entire facts before them, just as they are, in order that those who deserve credit may have it, and those who have enjoyed it without deserving it may be judged equally as to the merits of their work. The task is at best an unpleasant one, but it cannot be avoided by any one who is entrusted with the responsibilities of a scientific investigator connected with a matter of such vital importance to the welfare of this country as its great hog-growing interest, which represents an invested capital of \$220,811,082, which is incorporated in 44,346,525 animals, of which 2,334,525 are in Nebraska alone, the value of which is \$13,341,813. I say, that the scientist who neglects to tell the truth where such interests are at stake, is a poltroon unworthy the name of a man and undeserving of public confidence. It is, therefore, necessary to critically review Mr. Salmon's work from its beginning in 1878 to the present time. We will only state those points from the report of the former year which have an especial bearing upon the questions to be discussed.

First, as to the nature of swine plague, Mr. Salmon tells us that, "in my investigations of the contagious hog fever," p. 432, by which we must understand that he means the disease to be "contagious" in character. The next point of interest is, that thus early in his investigations Mr. Salmon asserted that there was but one porcine pest in this country, an opinion he held to until 1886, as previously stated. On this point he says:

"We may estimate the loss (of swine) in the entire state (North Carolina) at about 260,000 for the year ending April 1, 1878. * * In each of the counties named (see original) a considerable number of herds have been visited and examined, and, without exception, the living animals presented similar symptoms, and the dead ones showed similar changes in the different organs of the body. Slight variations were of course observed, as is always the case in any disease, but these were as great between individuals of the same herd, sick at the same time, as between different herds, even in different counties. And, what is of great importance, I did not find a single case in which it could possibly be supposed that death resulted from any local disease, but in every case a variety of organs, belonging to different apparatus, were found diseased."!! [The above language is of the

most positive character; it is the most absolute testimony that swine plague is a general disease, and as absolutely contradicts Mr. Salmon's latest assertion, that "swine plague" and "hog cholera" are two different and essentially local complications.] He continues: "The blood often showed marked changes; there were extravasations in various parts of the body, and always inflammation of the lungs and large intestines (!) [no specific local diseases are described in that language!], generally, also, of the heart, and often of the eyes; the skin, too, was plainly affected, and the temperature was found to be increased before any other symptoms of disease were present. * * * Considering all these facts, there can be no doubt that these animals all died of a general disease, a disease not caused by changes in any single organ, but a disease which caused the various organic changes observed." !! p. 435.

Again, he says:

"In every case the colon and cæcum were plainly affected, reddened externally, and internally showing changes varying from a simple coloration to inflammation and great thickening; in some cases they were studded with petechiæ, while in others there were none; ulcers of various sizes were frequently found, and also thickened, fibrous concentric patches.

"The cavity of the thorax, in every case, (!) contained a considerable quantity of turbid, bloody fluid. The pleuræ were generally thickened and covered with false membranes; the lungs were constantly found inflamed, occasionally in a few small spots only, but generally the greater part of the lung tissue was involved." p. 437.

It is necessary to again lay emphasis upon these two points. It will be observed that Mr. Salmon most unequivocally asserted, in 1878, that, "in every case, the colon and cæcum were plainly affected," and again, with equal positiveness, that "the lungs were constantly found inflamed, * * * generally the greater part of the lung tissue was involved."

Upon the question of the

"NATURE OF THE DISEASE,"

he asks the question,

"Is the affection a local or general one? In other words, does the disease originate from functional or organic disorder of any particular organ or apparatus, or are the anatomical lesions developed secondarily as the consequence of a general infection?

"This question, as regards the disease under consideration, can be answered in a definite and scientific manner. (!)

"Indeed, when we consider that the first symptom, and the one preceding the others by several days, at least, is an increase in temperature; that, when localized, a great variety of organs, belonging to different systems and apparatus, are involved—the lungs, pleuræ, bronchial tubes, heart, liver, stomach, intestines, spleen, kidneys, bladder, and skin; that there are considerable changes in the blood, as shown by imperfect coagulation, solution of the coloring matter, and blood extravasations, there can scarcely remain a shadow of doubt that the trouble is not a local but a general one." (!) 1878, p. 438.

As there is nothing communicated by Mr. Salmon to the report of 1879, we will now turn to that of 1880–81, and, in order to make the remarks still more pertinent, quote the opening language of that report upon swine diseases.

"SECOND REPORT OF D. E. SALMON.

"*Hon. Wm. G. LeDuc, Commissioner of Agriculture:*

"SIR—I have the honor to submit the following report of investigations undertaken by your authority.

"By the investigations carried out under your direction in 1878 many important and long-contested questions respecting this disease (swine plague) must be regarded as definitely settled. Among the most important of these, I particularize the following:

"1. The great epizootics among swine in the West and South are the result of one and the same disease.

[That is positive language! No talk about two diseases there!]

"2. The symptoms and more apparent lesions of this disease are definitely settled.

"3. This disease is contagious, and the great majority of cases may be traced to contagion." p. 13.

It is in the report in question that we find the first etiological investigations of Mr. Salmon recorded, and it is well to call attention to the fact that the report is entitled "Contagious Diseases of Domestic Animals, 1880–81."

The scientific investigations of this person may be divided into three etiological periods:

1. The micrococcus period, which is to be found in the reports from 1880–81 to 1884 inclusive.
2. The forged germ period, published in the report of 1885.
3. The doubtful period, published in the report of 1886, issued in 1887, in which swine plague is differentiated into two distinct diseases, viz.: "hog cholera," for which the forged swine plague germ of 1885

is retained, and "swine plague," for which Salmon has appropriated the true germ of the disease, first discovered by Dr. Detmers in 1878; rediscovered by me in 1886; seen by Klein in 1876, but not described correctly; discovered by me in the tissues of English swine in 1886; again discovered by M. M. Cornil and Chantemesse and Rietsch and his confrères in France, in 1887, of which later.

We will now proceed to discuss Mr. Salmon's "micrococcus period," in which he asserts that that organism is the sole and only cause of swine plague in the United States.

SALMON'S MICROCOCCUS PERIOD IN THE ETIOLOGY OF SWINE PLAGUE.

Under the heading, "Results of microscopic examination in regard to the nature of the virus," he says:

"Tube No. 1—pleural effusion—contained many very small granules—monococci; many couples of these—diplococci; and a few chains of three to ten elements similar in appearance to the single granules—streptococci."

Notwithstanding the differential nomenclature, these are all varieties of micrococci.

"Tube No. 4.—Blood. This being the only tube that could be filled with absolute certainty of excluding atmospheric germs, much more weight should be placed upon the results than either of the others.

"It contained considerable numbers of spherical granules similar to those found in the other tubes, and something less than 130,000 [!?!] of an inch in diameter. No bacilli, or cylindrical granules resembling their spores." pp. 20–21.

Salmon concluded his remarks, with regard to his experimental studies of the virus upon hogs, at this time as follows:

"1. The pig killed at Charlotte, N. C., July 2, 1880, was affected with swine plague, as is proved not only by its lesions, but by inoculations.

"2. The blood of this animal had not developed bacilli, even when preserved for six and ten days after slaughter.

"3. The microscope, with a power of one thousand diameters, revealed in the blood thus preserved vast numbers of spherical granules not all isolated as is seen in spore formation. * * * * but united in chains and clusters of every conceivable form, as occurs with micrococci, in active multiplication. In this blood could be discovered

neither bacillus rods, nor oval, or cylindrical spores of these. (See plate 1, fig. 1.)

"4. This blood was still virulent, as was shown by inoculation on animals, both of which sickened in seven days with the characteristic symptoms of swine plague; one of them when slaughtered presented typical lesions of this disease." p. 24.

The reader will observe that this was in 1880, some eight years ago.

Let us inquire a little more closely into the history of this "pig killed at Charlotte, N. C., July 2, 1880." Mr. Salmon tells us that,

"While at Charlotte, N. C., July 2, 1880, Mr. W—— informed me that hogs were dying of cholera on his farm near the city, and gave me permission to kill any animal that I might wish to examine. The one showing the most marked symptoms was selected and slaughtered. This animal had a large abscess in the flank; * * * similar though smaller ones extended beneath the thorax. The abdomen was distended with a colorless, transparent, peritoneal effusion; the intestines adhered closely from the formation of false membranes, and in the duodenum were many small erosions. The spleen was enlarged and the lymphatic glands engorged with blood. The pericardium contained one-half ounce of clear liquid; the lungs were mottled with lobular pneumonia, but there was no pleural effusion." p. 22.

Of the animals inoculated with the material upon which he bases the previous given conclusions, Mr. Salmon says:

"August 6th there were signs of improvement, when one of the animals was killed. The whole intestinal tract was found reddened and the mucous surface of the large intestine was studded with small ulcerations, the cæcum being most involved. The liver was mottled, and the lungs extensively hepatized. The blood before and after death contained spherical granules." p. 24.

Now it will be remembered that, of these results, Mr. Salmon says two very important things, viz.:

1st, That "the pig killed at Charlotte, N. C., was affected with swine plague."

2d, That "the blood of this animal had not developed bacilli rods, even when preserved for six or ten days after slaughter."

SALMON "STAMPS OUT" DR. DETMERS.

We must now call attention to the fact that Dr. Detmers had said in his reports of his investigations of swine plague (see 1878, 1879),

that an organism, to which he gave the name of "bacillus suis," was the cause of swine plague. In the report now under consideration (1880-81) Mr. Salmon makes his first endeavor to show that a "microoecus" and not a "baeillus" is the true cause of swine plague. It is also in this report that we hear the last of Dr. Detmers as a worker upon swine diseases for our agricultural department, though in the same he puts himself upon record as the first real discoverer of the true germ of the swine plague. To use a technical term, Mr. Salmon succeeded in "*stamping out*" Dr. Detmers and his work at this time, so far as these investigations went under the auspices of our Agricultural Department at Washington.

SOME CONTRADICTIONS.

"1. *Cultivations on Slides.* On March 10, five slides were prepared by putting a drop of fresh aqueous humor of a rabbit on a thin cover; this was then inoculated with the smallest possible particle of coagulum taken from a capillary tube filled at Pickens, S. C., Dec. 29, 1879, with effused liquid found in the peritoneal cavity of a pig suffering from swine plague. The cover thus prepared was then inverted over a Brunswick black cell painted on an ordinary glue slide."

The reader will be kind enough to notice the time which elapsed between the date at which this material was obtained, Dec. 29, 1879, and the day when this culture was started, March 10, 1880. Notwithstanding this interval, five hours later the drops of aqueous humor were swarming with single granules and clusters of these, nearly all with molecular motion; twenty-four hours after the preparations were filled with aggregations of granules, no movement in any and "no bacilli." p. 24.

"March 18. No bacilli have developed in any of these preparations; they have been examined carefully every day. In only one of them is there any activity; this swarms with single granules and small granulations, as in a freshly inoculated cell. Most of the clusters are of considerable size, held together by a gelatinous matrix. A few short rods $\frac{1}{2000}$ of an inch in length have been found. Nearly (! ?) the whole space to be seen is occupied by the granules.

"Final examination March 25. No material change since the last examination. All activity has ceased. The granules have not developed into filaments."

"March 12, four slides were prepared, using urine as a cultivating medium." In other respects the cultivation was the same.

"March 13 and 14 Micrococci alone are seen in various colonies as before."

"March 15, filaments have grown from a few clusters, but whether the granules of such clusters were identical with the others could not be ascertained; certainly the vast majority of clusters showed no signs of producing filaments." p. 25.

These quotations are sufficient to show that Mr. Salmon was not so certain as to his "granules" or "micrococci" as he would have the reader think. Bacilli or rods are certainly appearing in the very same cultures in which he tells us at the beginning of a passage, "March 10. No bacilli have developed in any of these preparations," while only four lines further on in the same passage, he says: "A very few rods $\frac{1}{12000}$ of an inch in length have been found." Again, in one place he says: "Nearly all have molecular motion," while in another he tells us, "no movement in any;" and in another, "all activity has ceased," and again, "in only one is there any activity."

Comment on the above is certainly unnecessary!

But to continue!

Further on, Salmon says:

"In regard to the parasite of swine plague, or, more particularly speaking, the form of organism found in the virus, Dr. Klein and Dr. Detmers believe it to be a bacillus, while Neguin (*Receuil de Med. Vet.*, 1880, p. 36) and myself have found granules which form clusters and chains but not rods." [As swine plague was not known as an independent disease in Germany until 1885, and in France until 1887, it is safe to assert that Neguin's studies may have been made upon either rouget or swine plague, neither of which is caused by "granules" or "micrococci."]

Salmon continues the defense of his "micrococcus" of this period as follows:

"Again, Dr. Klein could not have found bacilli in the virus, or he would not have considered the organism at first as a micrococcus. Dr. Detmers maintains that the rods (bacilli) exist in the blood and tissues even during life, while blood which I obtained by breaking capillary tubes within the blood vessels and immediately sealing them, developed chains of spherical granules alone and never bacilli. Even when such virus was cultivated on slides, I have only obtained the granules singly, in clusters, and in chains." p. 68.

Compare the last line of the above quotation with those previously given, where Salmon says: "A few very short rods have been

found;” “filaments have grown from a few clusters;” and then read the following :

“I was never able to obtain rods from the granules which I found in the virus; it seems to me the probabilities are entirely against the view that they are bacillus germs, or that they develop into a rod form.” p. 69.

Notwithstanding his attempt to show that Detmers and Klein were wrong up to this time, still Mr. Salmon has given no evidence for the correctness of his own assertions, even though he calls upon Neguin to support his tottering structure. His uncertainty is only too well shown by his contradictions, and, notwithstanding his endeavors to force Detmers into obscurity, he still shows he had done nothing to contribute to the etiology of swine plague, at this time, by the following :

“These are the results of my investigations of swine plague. I had hoped by cultivation experiments to prove that the granules observed either were the cause of the disease, or that they are an epiphenomenon, but owing to the fact that the virus, in every case, lost its activity after the first generation, or become too mild to afford satisfactory results, such evidences could not be obtained.” (Sic.) p. 69, 1880-81.

In order to render this testimony conclusive, I must be pardoned for again quoting what Mr. Salmon says on page 24 of the same report :

“1. The pig killed at Charlotte, N. C., was affected with swine plague, as is proved, not only by its lesions, but by inoculations made by Professor Law and myself.

“2. The blood of this animal had not developed bacilli even when preserved for six and ten days after slaughter.

“3. The microscope revealed in the blood vast numbers of spherical granules; in this blood could be discovered neither bacillus-rods, nor oval or cylindrical spores of these.

“4. This blood was still virulent, as shown by inoculation on two animals, both of which sickened in seven days, with the characteristic symptoms of swine plague, and one of which, when slaughtered, presented typical lesions of this disease.”

Such results as the last should be sufficient, and would be, did they emanate from any competent investigator, to demonstrate the fact that the “spherical granules,” “micrococci,” were the cause of the disease

in question. In this case, however, the author of them contradicts any such interpretation, when he tells us that "no such evidence was obtained."

Let us continue our researches upon this interesting question still further. For this purpose we must turn to the pages of the next annual report of our Department of Agriculture—that of 1883. Here we shall find a very singular "epiphenomenon." Notwithstanding Mr. Salmon had just told the country that, "I (he) had hoped by cultivation experiments to prove that the granules observed were the cause of the disease," and had admitted that "such evidence could not be obtained," still, without recording a single experiment, without giving another iota of positive evidence, we find him asserting in the most positive manner that a mitigated and artificial virus can be made from these very "granules" or cocci, in the following words:

"Our investigations have shown that the plague is a non-recurrent fever, and that the germs may be cultivated; they have even proved that these germs may be made to lose their virulent activities and produce a mild affection. Surely, we have here sufficient evidence to show that a reliable vaccine might be easily prepared if we carried our investigations but a little way farther."

It is now 1888; these investigations have been carried a big "little way farther"; the micrococcus, upon which the above assertions were made, has passed into "innocuous desuetude;" it has been completely "stamped out," and yet that promise of a "reliable vaccine" by the person in question, is a "little way farther" off from Washington than it was in 1883.

Mr. Salmon's next communication upon this subject is to be found in the Report of the Department of Agriculture, 1884, and entitled

"INVESTIGATIONS OF SWINE PLAGUE."

Having, as he thought, "stamped out" Dr. Detmers, Mr. Salmon now endeavors to show that he has claims for the original discovery of the germ of swine plague over both Dr. Klein, of England, and M. Pasteur, and still asserts that that germ is a micrococcus.

He begins as follows:

"In a communication of M. Pasteur to the Paris Academy of Sciences (*Comptes Rendus* 1883, p. 1163), it was asserted:

"1. That the microbe of swine plague is a dumb-bell micrococcus."

"To Pasteur's questions Dr. Klein (*Veterinary Journal*, London, 1884, July, p. 39) replies :

"1. That M. Pasteur has overlooked the true microbe, and that this is a bacillus and not a micrococcus." Report 1884, p. 221.

"That it is impossible to say whether M. Pasteur's rabbits died of swine fever or septicæmia, though he (Klein) had shown in 1877 that rabbits were susceptible to swine fever when inoculated from material derived directly from the pig."

"Dr. Klein does not hesitate to say that it seems probable to him that, 'as in the case of fowl cholera, M. Pasteur did not work with a pure cultivation of the germ of swine plague,' which is true, as Pasteur worked upon rouget, and swine plague was not known to exist in France until 1887, as we shall show later on. That Pasteur was also wrong as to a micrococcus being the cause of rouget, and that, even when he had produced an effective vaccine against that disease, he had no actual knowledge of its specific germ has been shown by Prof. Schütz, of Berlin. In the year 1885, the local government of Baden undertook the experimental testing of Pasteur's 'vaccine contra rouget,' the work being done by Pasteur's assistant, M. Cagny, but under the control of Messrs. Schottelius and Lydtin, two trustworthy German veterinarians. At the same time Prof. Schütz inaugurated a systematic investigation of the 'rouget,' or 'Rothlauf,' as it is called in Germany. He says, 'he received the spleen of a hog that had died from Rothlauf, from Lydtin, on the 7th of April, and in preparations from this spleen found great numbers of a very fine bacilli.' With pure cultures of this bacillus, Schütz inoculated mice, rabbits, and Guinea pigs, the results of which we need not here notice, and finally swine, and concludes his remarks as follows: 'These experiments conclusively prove that these bacilli sicken and finally kill swine. [Loeffler discovered this organism previously.] We now know that a disease occurs among the swine in Baden which is of a parasitic nature, the bacilli being identical with those found in the spleen sent by Lydtin.' *Archiv für Thier heil Kunde*. Berlin, 1885, p.p. 276, 285. It now becomes interesting to examine the vaccine fluid sent by Pasteur, which Schütz proceeded to do. Of this material he says: 'The vaccine sent me by Lydtin contained a mixture of micro-organisms aside from the fine bacilli.' Two white mice were inoculated with this material as received, and died on the 5th and 7th day afterwards, the fine bacilli being present in numbers." *Ibid.*, p. 367.

"Schütz then proceeded to isolate the organisms in the Pasteur vaccine in the artificial manner known to bacteriologists, that is, by 'plate-cultures.' The vaccine was found to contain six different organisms, among which was the fine 'bacillus.' Of the micro-organisms which Pasteur thought to be the cause of rouget, he, himself, says: 'Sur le Rouget ou mal rouge des pores.'—*Comptes Rendus*, 1882, p. 1120.

"Le mal rouge des pores est produit par un microbe special facilement cultivable en dehors du corps des animaux. * * * La forme est encore celle d'un 8 de chiffre, mais plus fin, moins visible que celui du cholera des poules."

The above shows that Pasteur had not seen the true germ of rouget, as a figure "8," or "dumb-bell micrococcus," is not a bacillus, or rod, by any means.

These investigations of Prof. Schütz first gave the key to rouget as an idiopathic malady of swine, (Loeffler's had not then been published, I think), while Detmers' work, 1880, had long ago shown that the American swine plague was caused by a very different organism, though the value of his observations, and all his claims for original discovery, had been successfully "stamped out" by the weight of authority of the later publications of Mr. Salmon.

Having shown how completely wrong M. Pasteur was as to rouget, and that he had no knowledge of the existence of any such disease as swine plague, proper, in France, we will now return to Mr. Salmon's work in 1884. He continues in his priority polemic against Klein and Pasteur, in favor of his "micrococcus" as the specific cause of our swine plague as follows:

"In a former report I (Salmon) have given details of experiments which, if correctly [why should Salmon doubt his own statement?] stated, demonstrate beyond all question that the microbe of swine plague is a micrococcus.

"These experiments were made, and the accounts of them published in advance of those of Pasteur, and the evidence furnished was all that could be reasonably required to decide a scientific question of this kind."

In the above passage Mr. Salmon claims priority over Pasteur, but this claim is completely nullified by the following, as Mr. Salmon's claims are dependent upon his work reported in 1880.

Klein says:

"In a report to the medical officer of the Local Government Board for 1877-78, I have shown that in this acute infectious disease the affected organs contain a bacterium in morphological respects identical with bacillus subtilis. * * * Last year Pasteur claimed to have cultivated from the blood of a pig affected with the disease a microbe which is not a bacillus, but a dumb-bell micrococcus." Micro-organisms and Disease, p. 94.

Now, by the context we must assume that by "last year," Klein means 1876, and if that be correct, Pasteur anticipated Salmon by four years in the discovery of a micrococcus.

Salmon continues :

"It is necessary, therefore, to review certain points in the investigations and to bring forward such new evidence as shall be required to remove these uncertainties."

The evidence is as follows :

"1. The microbe of swine plague. As I have shown elsewhere—*Science*, 1884, p. 155—Dr. Klein was the first to demonstrate the presence of micrococci in the tissue of animals that had suffered from swine plague, but he did not at that time—1876—attribute, nor has he since attributed, the cause of the disease to this organism.

"In my report of 1880 I have published experiments showing that the blood of sick and dead hogs, which had been received into vacuum tubes that were thrust inside the vein with proper precautions before being opened, contained micrococci, and no other organisms, and that hogs inoculated with this blood contracted a severe form of swine plague.

"This was the first discovery recorded, so far as I am aware, of the existence of micrococci in the blood of the affected swine before death." [That is true ! No one else has seen them !] "It has a very important bearing upon the etiology of the disease, since a post-mortem examination of the germs is out of the question, and they were found in situations to which there was no direct communication from the outside of the body.

"In my next report (1881-2, Department of Agriculture, pp. 206-7), I gave the details of experiments which demonstrated that these micrococci, after they had been carried through six cultivations in considerable quantities of liquid, were still capable of producing very marked cases of the disease known as swine plague."

Opposite page 224 of this report of 1884, we find a micro-photographic plate entitled 'Swine Plague Micrococcus,' to which Salmon refers in the following passage as Plate 11 (see Plate 1, Fig. 2 of this report and compare it with that of the micrococcus of 1880) :

"In the many cultivations which I have made from material obtained from slaughtered animals, I have never found bacilli except in a very few cases where the virus was not obtained until after contact with the air where the vacuum tubes had not been properly sealed, or where the animal was not slaughtered until the last stages of the disease.

"A photograph of a preparation made from one of these cultivations is reproduced in Plate 11.

"It seems to be a perfectly pure cultivation of micrococci, so far as careful examination with the microscope is able to determine, and it was so virulent that three pigs inoculated with it all contracted the disease and all died.

"In my most recent investigations I find that the peritoneal effusion is often impure in the last stages of the disease. In such cases a variety of organisms appear in the cultivations made with this liquid, but pure cultures of micrococci are still obtained from the pleural effusion.

"A fact of great importance is that no pure cultures of bacilli have been obtained, and that where but a single species of organism has multiplied, this has invariably been a micrococcus. (!!)

"Having obtained such results from my investigation, and having repeated them over and over again and confirmed them with virus from various parts of the country, I cannot but conclude that swine plague is due to a micrococcus, and that the disease produced by Dr. Klein's cultivated bacilli was a form of septicæmia."

What shall we conclude, who have read Salmon's latest productions? The above quotations from Salmon have, however, that appearance of exact evidence which should be "all that could reasonably be required to decide a scientific question of this kind."

Alas for Salmon's reputation! They have no value as evidence whatever. So far as the reputation of his micrococcus goes, he has knocked all support from under it.

But to continue with the report of 1884 and finish up with this part of the story. He says:

"These observations, which were made with great precautions to avoid errors, go far to reconcile the discrepancies which have appeared to exist in the results of the various investigations of this disease."

Will Salmon have the kindness to reconcile the discrepancies which exist in his own reports?

"The pericardial effusion contained large numbers of micrococci
* * * Cultivations gave pure growths of micrococci."

"Sections of the cecal ulcer contained enormous aggregations of
micrococci * * * no rods could be found."

"Pig No. 39, inoculated July 17th, slaughtered August 11th, presented well marked symptoms of swine plague. * * * Cultures of the pericardial fluid gave a pure growth of micrococci. Vacuum tubes filled with peritoneal fluid were preserved until September

2d, and were then found to contain large numbers of micrococci, but no other organisms."

"A large number of observations similar to the above have been made, and in all cases where a pure cultivation has been obtained the organism which multiplied was a micrococcus."

"When the virulence of such cultivated micrococci has been tested by inoculation experiments, typical cases of swine plague have resulted.

"Respectfully submitted,

"D. E. SALMON, D. V. M."

(Report of 1884, pp. 228-9.)

The above testimony is more than sufficient to show that from 1880 to 1885 Mr. Salmon considered a micrococcus to be the cause of our American swine plague.

We repeat but a few of his positive expressions. He tells us:

1. That "in all cases where pure cultivations have been made the organism that multiplied was a micrococcus."

2. That "when the virulence of such cultivated micrococci has been tested by inoculation experiments, typical cases of swine plague resulted."

3. That "the evidence furnished was all that could be reasonably required to decide a scientific question of this kind."

If the reader will turn to the reports of the department of agriculture for this period of four years he will find the numerous assertions that we have quoted from Mr. Salmon with regard to his micrococci being the cause of swine plague backed up by, apparently, the most positive kind of experimental evidence.

Let us see some of this evidence, which "was all that could be reasonably required to decide a scientific question of this kind." He says of some of them:

"The following record of experiments contains the most important of those which have been made since my last report, and is a continuation of the evidence upon which the above statements have been made:

"Experiment No. 1. Two pigs were inoculated June 28, 1883, with virus dried on quills and sent from Indiana. * *

The fourth day there was elevated temperature and a slight redness at the point of inoculation. The fifth day there was a diffuse redness on the inner side of both thighs, an eruption of small papule on the thin parts of the skin, and an increased elevation of temperature. *

* * The temperature reached its highest point on the 7th

(of July). From this time on they began to improve, and in neither case was the disease fatal." p. 226, Report 1884.

"This was one of a number of inoculation experiments made to obtain a reliable virus for experimental purposes, (!) and is recorded to illustrate the above remarks in regard to the period of incubation."

How that "reliable virus" was obtained from animals in which "*in neither case was the disease fatal*," Mr. Salmon fails to tell, but immediately proceeds with the details of another experiment in which other virus was used.

"Experiment No. 2. Four hogs were inoculated July 7, with virus also from Indiana, but obtained from an outbreak which was much more virulent and fatal."

"To and including July 17, or for the first ten days, there were small, hard swellings at the point of inoculation, but no positive signs of disease, and the appetite remained good. July 18, three were evidently sick. The one most severely affected was killed July 21, at which time the temperature was $104\frac{3}{5}^{\circ}$ F., and there was complete loss of appetite. The point of inoculation was much swollen, the enlargement extending forward under the abdomen, and was about six inches in length by two in breadth. When cut across it was found to be dense and fibrous and creaked under the knife (!) A clear lymph flowed from the cut surface. In the center of the swelling was an irregular cavity partly filled with dry caseous material. [These lesions produced in 14 days. Sic. !? B.] The right lung was nearly all of a deep red color, with extensive areas of infarction. There was a small quantity of effusion in the cavity of the thorax. The intestinal tract was congested, but there was no peritoneal effusion."

"The pleural effusion was collected in vacuum tubes with all known precautions. Cultivations were made with small quantities of this pleural effusion. All the attempted cultivations were successful, and the organism which multiplied was of identical appearance in each—it was a diplococcus, or figure eight in form, and had a tendency to adhere in short chains and small clusters."

"This organism was carried through three cultivations. August 2, experiment No. 3 was made by inoculating two pigs with the third cultivation of this micrococcus. This was made on the inner side of both thighs and with one between the fore legs also."

"There was swelling at the point of inoculation within twenty-four hours, but no marked increase of temperature until August 7, when it reached $105\frac{1}{5}^{\circ}$ with one, and $104\frac{2}{5}^{\circ}$ with the other, with impaired appetite, thirst, and shivering. Two days later the skin over the entire abdomen was wrinkled, flabby, and in places losing its epidermis. From this time they improved in general symptoms until

August 17, when the one which had received the larger quantity of virus was killed for examination."

"There was swelling of the lymphatic glands of the inguinal and mesenteric regions, petechiae of the serous membrane and slight peritoneal effusion." p. 227.

The "reliable virus" certainly could have had no relation to these animals, for both were killed, and there is no evidence that either would have died from the effects of the micrococcus.

But we have another experiment in which fatal results are reported to have followed the inoculation.

"Experiment No. 4. Three pigs, Nos. 26, 27, 28, were inoculated June 9, with a cultivation liquid seeded from the virulent effusion of a pig that had died from the result of inoculation with a very fatal virus received from Illinois." [It is singular that we find no records of the autopsy made upon such a very important animal!? B.] "This cultivation liquid contained only micrococci."

"June 14. All had elevated temperatures, varying from 104° to $105\frac{2}{5}^{\circ}$ F., increased thirst, tucked up abdomens, swellings at the point of inoculation, rigors, and secluded themselves in their bedding. The appetite was still fair."

"June 20. There was complete loss of appetite, emaciation, and profuse diarrhoea."

"June 29. No. 27 died, and the autopsy revealed congestion of the intestines, hepatization of the right lung, abundant effusion in the pleural, pericardial, and peritoneal cavities. Inoculations with this effusion caused the death of another pig July 8, after showing the well known symptoms of swine plague." [I desire to call attention to the fact of the incompleteness of these autopsy notes: the condition of the inside of the large intestines is not mentioned, but "hepatization of the right lung" is.]

"July 3. No. 28 was found in a dying condition, and was destroyed in order to get fresh material for examination and for inoculation experiments."

"July 6. No. 26 died in convulsions, after having presented the characteristic symptoms of swine plague."

"The notable point in this experiment is the virulence of the cultivated virus. This virus was a pure cultivation of micrococci, and produced fatal results in every case. The results of our inoculations with cultivated micrococci have heretofore been more or less unsatisfactory; because, while the symptoms were those of swine plague, the disease produced did not correspond in its malignancy to the swine plague which so frequently decimates the herds of the West." [The true reason is because "micrococci" have no etiological connection

with swine plague, as Mr. Salmon will very soon admit!] "In this case, however, the disease resulting from inoculation had all the malignancy of the most severe outbreaks which I have ever witnessed and in subsequent experiments with virus obtained from these animals, this fatal type has been retained and every animal inoculated has succumbed." pp. 227, 228.

Then follows the record of "pig No. 34, which was inoculated with mixed peritoneal and pleural effusion from No. 28. * * * July 18 it was very much debilitated, the breathing was rapid, and it was scarcely able to walk. It was killed for examination and for pure virus."

"Thoroughly sterilized vacuum tubes were filled from the jugular veins, from the right ventricle, and with the pericardial and peritoneal effusions. At the time of the autopsy small quantities of each of these effusions, and of the blood, were dried on cover glasses for examination in the laboratory."

"The tubes of peritoneal fluid, when opened, emitted a very disagreeable odor of putrefaction. Stained cover glass specimens showed that it contained micrococci and rods. Cultures of the same contained micrococci, a bacillus with pointed ends, and a few rods of bacterium termo. Cover glass preparations from the blood of the jugular vein presented no definite bacterial forms, even after staining. Cultures of this blood remained perfectly sterile. The pericardial effusion contained large numbers of micrococci. Cultivations gave pure growths of micrococci."(!) p. 228, 1884.

[Where did the micrococci come from? Under such circumstances they should have been in the blood, from which "covering glass were prepared at the time of the autopsy"!]]

Mr. Salmon concludes his work of that year as follows:

"A large number of similar observations to the above (!) have been made, and in all cases, where a pure cultivation has been obtained, the organism which multiplied was a micrococcus, and when the virulence of such cultivated micrococci has been tested by inoculation experiments, typical and fatal cases of swine plague have resulted." p. 229.

The reader will observe that Mr. Salmon says, "in all cases where a pure cultivation has been obtained," and yet on page 228 of the same report he had told us that, "the results of our inoculation experiments with cultivated micrococci, have been more or less unsatisfactory, * * * because the disease produced did not correspond

in its malignancy to the swine plague which so frequently decimates the herds of the West." Furthermore, we have seen from his own experiments that a large number of his experiments gave uncertain results.

MR. SALMON DISCOVERS A "NEW MICROBE" AS THE CAUSE OF
SWINE PLAGUE.

The next annual report of Mr. Salmon is that of 1885, but which was issued in the summer of 1886, and came into my hands after I had begun my own investigations. It is necessary, in this place, for me to say, that when I came to Nebraska, and up to the time that I began my personal researches into the cause and nature of swine plague, I was somewhat, though not critically, familiar with the work reported in the Reports of the Department of Agriculture upon the disease. Dr. Detmers' work had been entirely driven from my mind, and only that of Mr. Salmon had made any firm imprint on my memory. At that time, had anybody asked me what organism was the cause of swine plague in this country, I should have unhesitatingly answered a "micrococcus," and referred to Mr. Salmon's work as evidence. European investigators had quoted his assertions against Detmers, but seem now to have entirely forgotten that they had done so. Notwithstanding the fact that the report of Loeffler and Schütz's discovery of the germ of a swine plague in Germany came into my hands before I went to Europe with the somewhat notorious "Newark boys," in which they demonstrated that another organism than a bacillus was the cause of that disease, and hence permanently differentiated it from the rouget, or erysipelas, still I had no idea that it was an organism of the same species which caused our American swine plague. I therefore commenced my work with the best faith in Salmon's "micrococcus." Before receiving his report of 1885 (in the summer of 1886), I had had my faith shaken in his authority by the discovery of apparently the same germ as Schütz described in a very large number of diseased hogs, and could not possibly find a "micrococcus," and never have been able to find one, for the simple reason that it was not to be found in and never existed as the cause of swine plague in this or any other country. Still my surprise was great, on opening Mr. Salmon's report of 1885, to find the following:

"Anticipating somewhat the conclusions which we arrived at later, concerning this puzzling disease, we must say at this point that we no

longer consider a micrococcus to be the cause of all the outbreaks of the disease known as swine plague." p. 186, report of 1885.

What then is the scientific world to say as regards testimony which was "all that could reasonably be required to decide a scientific question of this kind"?

What shall we say when we compare the above quotation from his report of 1885 with the following from 1884:

"1. In all cases where pure cultivations have been made, the organism that multiplied was a micrococcus."

Mind you, Salmon says, "in all cases." Do not mistake the words, "in all cases." For these four years a micrococcus was the cause of all the outbreaks of swine plague.

When he wrote later that it was not the cause of all outbreaks of swine plague, he certainly must have entirely forgotten that only the year before he had written:

"2. When the virulence of such cultivated micrococci has been tested by inoculation experiments, typical cases of swine plague resulted."

"Typical cases!" Nothing could be more positive.

What kind of organism, then, did Mr. Salmon describe as the specific cause of our swine plague in 1885?

We will let him tell us.

"THE BACTERIUM OF SWINE PLAGUE.

"In at least twenty-five cases of undoubted swine plague, bits of spleen tissue, when spread out in a thin layer on a cover glass, dried and stained in some aniline color, were found to contain the same microbe in greater or less abundance (Plate II., Fig. 1). [Plate I., Fig. 3, of this report.] When stained for from one to two minutes in an aqueous solution of methyl-violet and examined with a Zeiss $\frac{1}{18}$ homog., they appear as elongated ovals, chiefly in pairs. The greater number present a center paler than the periphery. This may be due to a greater density or straining capacity of the peripheral portion. The darker portion is not localized at two extremities as in the bacteria of septicæmia in rabbits, but is of uniform width around the entire circumference of the oval. The length of an oval in balsam preparations is about 1.2 to 1.5 micromillimeter; its width .6 micromillimeter." Page 212, Report 1885.

The above statement is supported by as apparently conclusive testimony, of an experimental nature, as was the micrococcus of former years, some of which we now append.

Before quoting it, however, it is necessary to call the reader's attention to the fact that these experiments lose their entire value as positive testimony to the genuineness of this "microbe" as the cause of swine plague, because they had an "outbreak at the station," and the text itself shows that the animals that died were far more likely to have been infected from local causes than from artificial interference. The test pigs often died before or the same day of the inoculated ones, or the latter died unexpectedly. Furthermore, their value is absolutely nullified for the simple reason that no such germ exists in connection with swine plague as that described above. This testimony is therefore valueless, and is simply introduced to show how they work at Washington. There is no question that the animals died of swine plague, however, if they died at all.

Mr. Salmon continues:

"CONCLUSION OF THE INVESTIGATIONS CONCERNING THE CAUSE OF
AMERICAN SWINE PLAGUE.

"The two animals which infected the vaccinated pigs, as described in the preceding pages, deserve our attention more particularly, since they were the starting point of an outbreak at the experimental station, which has finally enabled us to demonstrate as the cause of the disease a specific microbe. This outbreak was characterized by great virulence, and most of the infected animals died in the early stages of the disease.

"These two animals, when brought to the station November 4, exhibited the usual symptoms of swine plague, great depression, with profuse diarrhoea. The owner stated that they had been sick for about a week. On the following day one (No. 105) was so low (temperature 95°F.) that we decided to kill it, the warm weather not promising good preservation if it should die in the night. It was killed by a blow on the head. The skin was slightly bluish in the axilla, a similar but less marked discoloration on the abdomen. The superficial inguinal glands were greatly enlarged, the individual lobules standing out prominently, some of a pale flesh color, others purplish, medulla pale. In opening the abdomen a few whitish patches were observed on the small intestine, corresponding to ulcerations, as determined later.

"There was a moderate quantity of watery serum in both pleural sacs. The lungs were normal, with the exception of a small anterior

lobe on each side which was hepatized. The pericardium was slightly distended with a colorless fluid, a small clot in each ventricle. Very severe lesions were found in the intestinal tract. The partially empty stomach contained two coiled up specimens of *ascaris*. The pale mucosa was studded with several isolated yellowish ulcers, about one-fourth of an inch in diameter, raised above the surface and flattened at the top. In the ileum extensive ulcerations were found, extending for a distance of about two feet from the valve. These ulcers had a depressed base, as if the tissue had been dug away, and were surrounded by a smooth elevated border. In the cæcum and large intestine in general, the ulcerations were very numerous, varying from one-eighth to one-third of an inch in diameter. The smallest ones appeared as yellowish specks. The largest ones were slightly depressed, containing black, ragged, necrotic masses. The lymphatics, at the root of the mesentery and near the ileocecal valve, were greatly enlarged, representing a continuous cylindrical mass, at least an inch thick, and varying from a pale flesh color to a dark red.

"This was evidently a severe case of swine plague, and one which, from previous experience, would prove unsatisfactory for purposes of investigation. Three cover-glass preparations from the spleen, one from the liver, two each from the hepatized lung tissue, and blood from the heart, were searched with negative results. *No bacteria could be seen.* A culture in a tube of nutritive gelatine rapidly liquefied the gelatine in the track of the needle.

"Liquid cultures were made by inoculating sterile nutritive media with a platinum wire dipped into the parenchyma of the spleen and liver, exposed by a cut with a flamed knife. Both contained a motile bacterium, identified later as the bacterium of swine plague. When line cultures were made on gelatine, that of spleen was obviously pure; the colonies from the culture of liver were of two kinds—one, the bacterium of swine plague proper, as determined later; the other growing in colonies having only one-fifth the linear dimensions of the former. A liquid culture, prepared by rubbing the platinum wire over the peritoneum, contained, when tested by the above method, the swine plague bacterium." p. 194-5.

"Of the four inoculations with pure cultures of the bacterium of swine plague to be described further on, the first did not prove quite satisfactory, from the fact that the cheek-animal died before either of the inoculated animals. [What killed it?] It will be seen, however, that this need not necessarily be considered as militating against our interpretation. The autopsy showed complete necrosis of the mucous membrane of the cæcum and colon for a distance of 2 feet. Beyond this the membrane was dotted with isolated ulcers. These ulcers were so deep that the serous membrane became inflamed. On each thigh at the seat of inoculation a large whitish mass was found over 2.5 inches long and .5 inch thick. The spleen of this animal contained

but a few bacteria. No cultures were made, as subsequent inoculation experiments had already furnished satisfactory results.

"No. 109 lingered until January 7, 1886, when it was killed. In this animal tough tumors were found at the point of inoculation larger than a hen's egg. The lungs were more or less affected, but the presence of lung-worms leaves the cause of the lesions a matter of doubt. In the cæcum, however, there were extensive ulcerations, very deep, implicating the serous covering and producing inflammatory adhesions with the rectum. The lymphatic glands were enlarged, but pale and tough. The spleen contained no bacteria; a liquid culture therefrom remained sterile.

"The healthy cheek-pig (No. 110) penned with these died December 6, after four or five days of illness, during which period the feces were at times covered with blood. [The same cause that killed 110 killed the others.—B.]

"From the foregoing description we observe that the cheek-animal died from a very acute attack, and it seems reasonable to suppose that it caught the disease from the two inoculated animals, and, being the more susceptible, quickly succumbed to the virus. [How about the pigs that introduced the plague to the station? They seem to have passed into "innocuous" forgetfulness, though they left active results behind.—B.] The herd from which this animal was taken did not allow any suspicion as to its soundness, considering later observations. It is highly probable that the disease appeared early in the inoculated animals, but owing to its comparative mildness remained unnoticed for a time. The cultures from No. 110 were successful in demonstrating the presence of the oval motile bacterium. Compare with this the negative results of the chronic cases. pp. 198-199.

"Among those cases in which swine plague was definitely made out on post-mortem examination, may be mentioned No. 88, which was exposed to the disease Nov. 5, and died Nov. 16. Covering glass preparations of the spleen revealed the presence of numerous microbes, slightly longer than the bacterium of swine plague and without the light-centers. * * * Liquid cultures from the blood, when tested by live cultures, showed the characteristic growth of the bacterium of swine plague, but there were, in addition to these, a few smaller colonies, growing like them, so few in number, however, that they were regarded as retarded colonies of the same microbe, their small size excluding a microscopic examination." p. 210.

Query: What became of the "slightly longer microbes" from the "covering-glass preparations of the spleen?"

"In case of No. 99, the temperature likewise rose to $107\frac{1}{2}^{\circ}$ F. on November 30. It died on December 7. The lesions resembled those of No. 98, with the following exceptions: In the left side of the

abdominal cavity a large clot of blood was found beneath the peritoneum, extending from near the diaphragm into the pelvis and representing probably 300^{cc} of blood. The left kidney was entirely imbedded in it. The place of rupture could not be found, owing to the firmness of the clot. Both kidneys pale. Glands of the intestinal tract prominent but very pale. In the stomach the food there present was encased in a dark coagulum. The hemorrhage probably came from the base of the folds at the fundus, where the mucosa was very dark red. In the cæcum and large intestine the mucous membrane was studded with jet-black pigment patches collected into lines and groups. The valve was covered with these ecchymoses showing signs of ulceration. This condition prevailed throughout the large intestine; the rectum seemed intact. Echinorhynchii in small intestine. The oval bacterium found on cover-glass preparations of the spleen in moderate quantity. Two cultures in tubes containing gelatine were prepared, one from blood taken from the heart, the other from the spleen. In forty-eight hours a small number of whitish points were present in the blood culture. In that of the spleen, however, each needle track contained a large number of these minute colonies. In addition to these there were, in all, five colonies distinguished from the rest by their large size. Two cultures in meat infusion peptone inoculated with blood from the heart, when tested on gelatine plates, were found pure. A liquid culture from the spleen gave different results. The line on the gelatine plate along which the bacteria had been sown was visible as a white line in twenty-four hours, while the colonies of the bacterium of swine plague do not appear within forty-eight hours after sowing. The surface growth especially was quite vigorous, enlarging within three to four days into an irregular whitish band. The microbe resembled that of swine plague, but was larger and stained more deeply. (!) The liquid culture itself, when re-examined, was covered by a brittle membrane. The tube culture in gelatine demonstrated that the strange microbe was present in very small numbers in the spleen itself. This illustrates very clearly how one method of culture acts as a check on the other, and how each contributes something to the determination of the truth. The presence of another organism in the spleen need not be very surprising when we consider the severe hemorrhage mentioned above." p. 207—1885.

"On December 5, 2 pigs (Nos. 121, 140) were inoculated with a pure liquid culture, about seven days old, from a superficial inguinal gland of pig No. 97, each receiving about 3½^{cc} of the culture liquid. On December 10, the temperature of No. 121 rose to 107½° F. It was found dead quite unexpectedly (!!) on the morning of December 12, having eaten heartily the evening previous.

"In this case the kidneys and lungs seem to have suffered most, if we exclude the lymphatic system. [No ulceration present—in the intestines!]

"Cover-glass preparations of the spleen showed the oval bacterium in large numbers. In two liquid cultures of the spleen the motile bacterium only was present. A gelatine culture of the spleen showed in each needle track the innumerable minute colonies of the same bacterium.

"No 140 had a temperature of $107\frac{1}{2}^{\circ}$ F., December 10. Eyes inflamed. It died December 18 quite unexpectedly (!) after having improved slightly a few days previous. The autopsy revealed a very severe case of swine plague. It differed from the preceding case in the presence of extensive ulcers of the large intestine, accompanied by similar ulceration of the ileum for about 2 feet from the ileo-caecal valve. The lungs were congested and hepatized anteriorly. The kidneys and lymphatic glands, generally, were also involved. Cover-glass preparations of the spleen contained the characteristic bacterium. A gelatine tube culture from the same organ gave the characteristic minute colonies in large numbers. The cheek-pig (No. 122) placed with this pair on the day of inoculation died on the same day with No. 140. [Pen infected?] It had probably (!) been infected by No. 121, as it had succumbed very suddenly, and the autopsy revealed a very acute case of swine plague of the hemorrhagic type. Lungs, intestines, and lymphatic glands were severely diseased; ulcers had not yet formed. The spleen contained the oval bacterium in abundance, as shown by cover-glass preparations and a culture in gelatine." p.p. 205-6.

"On November 27 two pigs (Nos. 111, 114) were inoculated by a subcutaneous injection of a liquid culture made from the spleen of pig No. 96, and found to consist of only one kind of microbe, when tested by line cultures on gelatine plates. The microbe was motile, grew on gelatine like that isolated from previous cases, and was presumably (!) the microbe of swine plague. On December 5 the temperature of one of the animals (No. 114) was $103\frac{3}{8}^{\circ}$ F. Bowels somewhat loose at first. Appetite good until death, which took place between 9 and 12 A.M., December 6, about nine days after inoculation.

"The examination was made December 7, a temperature below freezing preventing post-mortem changes. The superficial inguinal glands were found considerably swollen, the section dotted with red points and lines. The spleen was somewhat enlarged and darker than normal. The right heart distended with dark, imperfectly coagulated blood. On both auricular appendages a number of well-marked extravasations, some the size of a pin's head; on endocardium of left heart a few, not larger than mere points. Lungs oedematous and of a pale reddish hue, especially marked along the edges of the lobes. Bronchial glands enlarged, of a dark red color throughout. The glands at the root of the mesentery and about stomach very large and confluent, of a mottled appearance. On section the medulla hyperæmic. On tearing apart the coils of the large intestine the

glands of the meso-colon appeared as purplish red bean-shaped bodies gorged with dark blood. Beneath the serous coat of the cæcum at its very tip were numerous punctiform extravasations. The kidneys were severely inflamed. On the surface of both, numerous punctiform extravasations. On section the pyramids, including the tips of the papillæ, of a dark red color. The cortical portion dotted with innumerable dark red points. The lymphatic glands in the abdomen itself were of the same purplish color. On examining the mucous membrane of the intestinal tract, a large, deeply reddened patch of mucous membrane was found on the greater curvature of the stomach. The small intestine seemed intact excepting near the ileo-cæcal valve, where the longitudinal folds were of a dusky red, brought about by aggregations of minute dark red points. A similar condition prevailed throughout the large intestine, giving the entire surface a dark appearance. In many places small, blackish ecchymoses indicated hemorrhages on the surface. The kidneys in this animal seemed to have suffered most severely, next to the large intestine, which in all our examination was obviously the seat of the severest lesions.

"Cover-glass preparations of the spleen, kidney, and liver, examined immediately, revealed the same microbe which had been introduced into the system.

"Cultures in tubes of gelatine from blood of the heart and spleen resembled precisely the pure cultures from previous cases. As usual, the colonies in the culture of the blood were few and scattered; those in the spleen culture were innumerable in each needle track. (Plate II., Fig. 3.) Two liquid cultures inoculated with blood from the heart and one from the spleen were tested as above and found pure, containing only the motile bacterium.

"The second pig (No. 112) [above "No. 111—which?"] inoculated with the same culture and in the same manner as No. 114, died in the morning of December 12. The temperature first rose to $105\frac{3}{4}^{\circ}$ F. on December 8; diarrhœa set in on December 10. It was found dead on the morning of December 12. The lesions closely resembled those of No. 114 with the following differences: The spleen was much enlarged, gorged with blood, and very friable. On one border there were prominent red points, giving it a ragged appearance. Numerous ecchymoses beneath the endocardium of the left ventricle. On the surface of the lungs numerous dark red spots corresponding to hepatized lobules. These, about one-fourth of an inch in diameter, were found throughout the lung tissue. The kidneys, though congested, were not so seriously affected as in No. 114. For about 4 inches from the valve the summits of the folds of the mucosa of ileum were deep red, consisting of aggregations of red points. A large patch of mucosa in the cæcum purplish. The large intestine, in general, congested and covered with dark red points. No ulceration. (!) The enlargement of the lymphatics and inflammation of the stomach, as in

preceding case, but more severe, the mneosa of the greater portion having almost a black color, due to extravasation. Cover-glass preparations of the spleen contained but few specimens of the bacterium of swine plague. After inoculating Nos. 112 and 114, a third healthy check-pig, No. 111, (!?) [inoculated above] was placed in the same pen. This animal remained well for two weeks after the death of the second animal, when it began to show signs of disease. It died December 31, more than a month after being penned with Nos. 112 and 114. The autopsy demonstrated swine plague with extensive ulcerations of the large intestine and implication of the lymphatic glands, lungs, and stomach. No bacteria were found in three cover-glass preparations of the spleen. This animal, therefore, remained well until infected by the two inoculated ones. It is evident that no check-animal will remain ultimately intact, owing to the manner of infection, and hence this must not be looked for in experiments with swine plague." pp. 201-2—1885.

How about contagion, Mr. Salmon? This case seems to completely "stamp out" that contagious theory!

From these results Mr. Salmon came to the following:

"CONCLUSIONS."

"The preceding investigations definitely settle certain controverted points concerning the etiology of swine plague, which may be briefly summarized:

"(1) Swine plague is caused by a specific microbe multiplying in the body of the diseased animal. The microbe probably belongs to the genus bacterium, and has the power of spontaneous movement. It is easily cultivated in nutritive liquids, but grows less readily on gelatine, which it does not liquefy.

"(2) When introduced beneath the skin, this bacterium is fatal to pigs, rabbits, guinea-pigs, mice, and a certain percentage of pigeons. It is also fatal to pigs when introduced with the food or when they feed on the internal organs of swine which have died of the disease.

"(3) The disease described in France as *rouget*, in Germany as *Rothlauf*, and for which Pasteur has prepared a vaccine, is caused by an entirely different microbe. The vaccine for this disease does not protect against swine plague.

"(4) The introduction of Pasteur's vaccine is not only useless, but may contribute to the introduction and spread of a disease, the existence of which in this country has not yet been demonstrated." p. 229, 1885.

COMPARISON OF MR. SALMON'S ASSERTIONS AS TO THE CAUSE OF
SWINE PLAGUE FROM 1880 TO 1885.

SALMON'S CONCLUSION AS TO THE
NATURE OF THE CAUSE OF HOG
CHOLERA.—1880-84.

"The blood of pig killed at Charlotte, N. C., July 6, 1880, which was an undoubted case of swine plague, had not developed bacilli, even when preserved for six and ten days after slaughter."

"The microscopic examination of this blood revealed vast numbers of spherical granules—micrococci—and in this blood could be discovered neither bacillus rods, nor oval, or cylindrical spores of them."

"Inoculations of animals—pigs—with this blood, caused them to sicken, in seven days, with the characteristic symptoms of swine plague; one of them, when slaughtered, presented the typical lesions of this disease."

SALMON'S CONCLUSIONS AS TO
THE NATURE OF HOG CHOL-
ERA.—1885.

"Anticipating, somewhat, the conclusions which we arrive at later, concerning the real cause of this puzzling disease, we must say at this point, that we no longer consider a micrococcus as the cause of all outbreaks of the disease known as swine plague." p. 186.

"The first annual report of the bureau, 1884, mentions the death of three pigs from inoculation with a micrococcus, * * *

One of the animals was killed about seventeen days after the inoculation. Lungs found to be extensively hepatized—solidified. The presence of numerous lung worms left no doubt as to the cause of the hepatization. Tubes inoculated from the spleen and blood remain sterile. The second animal died of swine plague one month after inoculation." p. 186.

"The results which we obtained, later on, with another microbe, led us to suspect that this was a case produced by natural infection." p. 186.

Salmon will please tell us if all the cases of inoculated swine plague which he asserts were due to his micrococcus, before this date, and which he quotes as "all the evidence which could reasonably be required to decide a scientific question of this kind" were also "produced by natural infection."

"Whether this micrococcus is a

septic organism or one which is the cause of a definite disease in pigs cannot be answered at present." p. 186.

1883—"M. Pasteur has recently confirmed our American investigations in a very complete manner. He shows that the disease is produced by a micrococcus."

1884—"In a former report I have given the details of experiments which * * * demonstrate that the microbe of swine plague is a micrococcus, and the evidence furnished was all that could be reasonably required to decide a scientific question of this kind."

"This was the first discovery—report of 1881—recorded * * * of the existence of micrococci in the blood of the affected swine before death."

"I have given details of experiments which demonstrated that these micrococci, even after they had been carried through six cultivations * * * were still capable of producing swine plague."

"This I believe was the first satisfactory evidence of the pathogenic effect of the micrococci in the disease known as swine plague."

"I cannot but conclude that swine plague is due to a micrococcus."

"This virus was a pure cultivation of micrococci, and produced fatal results—the swine plague—in every case."

"A large number of observations, similar to the above, have been made, and in all cases where a pure cultivation has been obtained, the organism which multi-

"THE BACTERIUM OF SWINE PLAGUE.

"In at least twenty-five cases of undoubted swine plague, bits of spleen tissue, when spread out in a thin layer on a cover glass, dried and stained in some aniline color, were found to contain the same microbe in greater or less abundance. (Plate III., Fig. 1.) When stained for from one to two minutes in an aqueous solution of methyl-violet and examined with a Zeiss $\frac{1}{8}$ homogenous, they appear as elongated ovals chiefly in pairs. The great number present a center paler than the periphery. This may be due to a greater density or staining capacity of the peripheral portion. The darker portion is not localized at the two extremities as in the bacteria of septicæmia in rabbits, but it is of uniform width around the entire circumference of the oval." p. 212.

"The earlier work, recorded in the preceding pages, aimed to determine what relation the bacillus cultivated by Pasteur as a vaccine for rouget, bore to the disease among swine prevailing in this country. At that time—Oct., Nov., 1885—the bacterium of swine plague had not been seen by us."! p. 217.

See opposite column!

"The bacterium which we have lately discovered and which we believe to be the cause of swine plague." p. 219.

plied was a micrococcus, and when the virulence of such cultivated micrococci has been tested by inoculation experiments, typical and fatal cases of swine plague have resulted."

Respectfully submitted,
D. E. SALMON, D. V. M.

He is not quite sure yet!

"Up to this time (Oct., Nov., 1885), we had not obtained uniform results pointing definitely to any microbe as the cause of swine plague." p. 230.

"Micrococci Salmon! 1880-85."

"The kidney of the third one—No. 4—contained large numbers of the oval bacteria found in cover-glass preparations of the spleen of pigs affected with swine plague later on, and identified as the cause of swine plague."

"This was the first time these bacteria were seen." p 196.

"CONCLUSIONS.

"The preceding investigations definitely settle (!!! ??? B.) certain controverted points concerning the etiology—cause—of swine plague, which may be briefly summarized:"

1—"Swine plague is caused by a specific microbe multiplying in the body of the diseased animal. This microbe, probably, (1) belongs to the genus bacterium *

* * —not coecus—this time."

2—"When introduced beneath the skin, this bacterium is fatal to pigs, rabbits, guinea pigs, mice, and a certain percentage of pigeons. It is also fatal to pigs when introduced with the food, or when they feed on the internal organs of swine, which have died of the disease." p. 229. So was the micrococcus of 1880-85.

The above remarks were printed in several papers in October, 1886, and called forth the following reply in the "Breeders' Gazette" of November 11, 1886:

TWO DIFFERENT GERMS FOUND IN HOG CHOLERA.

"The publication by Dr. Billings in at least three different journals of several columns of exaggerated criticisms, and unfounded statements, based upon alleged discrepancies in the published results of our microscopical work in connection with the hog cholera [It was 'swine plague' then, and had reference to the work from 1878 on. B.] for the years 1884 and 1885 leads me to make an announcement in regard to recent investigations in advance of the publication of my report.

"It will be remembered that in 1884 I attributed the cause of hog cholera to 'motionless micrococci of the figure-of-eight form' (p. 225), and gave a photograph of a culture of these, which was 'so virulent that three pigs inoculated with it all contracted the disease and all died.' A reference to this photograph, which faces page 224 of the report for that year, shows that the description was correct, and that the germ there illustrated resembles very closely the drawing of the organism of fowl cholera published in the 'Annual Report of the Department of Agriculture for 1881 and 1882' (Plate VII.) (Fig. 2, Plate I.) I refer to this because the fowl cholera microbe has generally been called a micrococcus in my own writings, and I think also in the writings of a majority of biologists. Some recent German authorities, however, refer to this germ as a bacillus, and others call it a bacterium. This simply illustrates how the names of this class of organisms differ according to our ideas of classification. The fowl cholera germ occupies a position between the micrococci and bacilli, resembling both and differing from both. If I were writing of it at present I should follow those who class it in the genus bacterium, but only four years ago I placed it in the genus micrococcus.

"This change of ideas about classification, however, does not greatly affect the practical results of investigations, since if a description and drawing is made of a germ we recognize the thing referred to whether it is called a micrococcus or a bacillus. I mention this simply to show that in 1884 I gave a photograph of a motionless figure-of-eight organism obtained from an animal affected with swine plague, which when inoculated was fatal to pigs, and I concluded, very naturally, I think, that this was the cause of swine plague.

"Very soon after these experiments were made, the many duties which devolve upon me as chief of the Bureau of Animal Industry made it necessary to place the investigations of hog cholera almost entirely in the hands of my assistant, Dr. Theobald Smith, in whose ability and devotion to the work I had and still have the utmost confidence. While therefore I have marked out the lines of investigation and kept a personal supervision over what was done, the work has been carried out by another.

"Now one of the very first results of this arrangement was the

conclusion by Dr. Smith that hog cholera was caused by a motile bacterium, which certainly was a different germ from the one that I had described in 1884. I looked over his preparations, made sure his experiments were properly carried out, and that the disease produced by inoculation and cohabitation presented the symptoms and lesions which are seen in the swine plague, and then published his results almost in the exact language in which he wrote them. Among other slight changes which I made was this: a phrase which read 'we no longer consider a micrococcus as the cause of swine plague,' was changed to 'we no longer consider a micrococcus as the cause of all outbreaks of the disease known as swine plague.' p. 186.

"In publishing all the facts which came under my observation, and suppressing none, I believe I was following the dictates of true science, even though there appeared to be discrepancies which might be construed to bear against the value of my own work. If I had made a mistake I certainly could point to many excellent men who had made equally bad mistakes in pursuing this difficult line of investigations, and if my conclusions were in any degree correct the future would certainly bring demonstrations of it. I am now in a position to explain the supposed discrepancy to the satisfaction of any reasonable person.

"Several months ago we obtained from an animal supposed to be affected with hog cholera, but which had peculiar lesions of the lung, a figure-of-eight organism which was non-motile in liquid cultures. This germ we have since found in other outbreaks of swine disease, and we are making a thorough study of it and of its relations to the epizootics in various parts of the country. The microscopical characters of this germ, so far as we have studied it, are identical with those of the fowl cholera microbe, and I believe it to be the same germ which I described in my report of 1884.

"There is an error in the report of 1885 (p. 186), where the statement is made that this germ rapidly liquefies gelatine. This error was the result of a mistake which occurred when the laboratory work was transferred to Dr. Smith. I had a number of cultures of micrococci of different species which had been obtained from diseased hogs, and in some way a culture of non-virulent germs was mistaken for the virulent species photographed in the report of 1884. As a result the virulent germ was lost for the time, and we found ourselves in possession of a micrococcus which liquefied gelatine, but which did not produce any symptoms of disease in any species of animal. The germ described in 1884 had been found very fatal to both pigeons and pigs. The misstatement referred to, which naturally resulted from this error, was not discovered in time for correction in the report.

"This microbe, which I then called a micrococcus, but which, according to my present ideas of classification, would be more correctly termed a bacterium, was not encountered again in an outbreak of

swine disease until the present summer. It is what has been called a figure-8 organism, which stains at its extremities, and appears to be identical in microscopical character with the microbe of rabbit septicæmia, with that of fowl cholera, and with that described by Schütz as the cause of the German disease called *schweineseuche*.

"This bacterium has been obtained by us from the pleural effusion, the blood, and the spleen of diseased pigs. It is fatal to mice, rabbits, guinea pigs, pigeons, fowls, and hogs. It differs from the bacterium of hog cholera, described in the report of 1885, in the parts of the figure-8 form being more nearly spherical and in staining at the extremities instead of uniformly around the whole periphery; it is non-motile in liquid cultures, while the other is motile; the colonies on gelatine are different; we have not been able to cultivate it on a potato, a situation very favorable to the other germ; rabbits live only three to five days after inoculation, while with the other germs they live seven to eight days; mice live only two to four days, while with the other they live eight to sixteen days. It is very fatal to fowls, in which the other germ produces no serious results.

"We hope to continue our investigations of this germ until we are able to say definitely what part it plays in the losses of swine which have heretofore been grouped together as caused by one disease.

"I trust that this statement will satisfy those interested in this matter that the scientific work of the Bureau of Animal Industry has been faithfully and honestly performed; that there are no serious discrepancies in the reports of the last two years, and finally that we are making as much progress as can be expected with so complicated a disease, and in a laboratory where the investigations cannot be pushed beyond a certain limit for want of space to work.

"I am glad to see that Dr. Billings has confirmed my work of 1884 by stating that he has discovered in Nebraska a germ which 'exactly corresponds in its microscopical appearances to that discovered and described by Schütz of Berlin, in the *schweineseuche* of Germany.' After a few years' experience in this class of investigations he may learn, however, that he has made a mistake in jumping to the conclusion that this is the only pathogenic germ found in the diseases which have been, up to this time, classed together in this country as hog cholera.

"D. E. SALMON."

The previous communication called forth the following reply:

It was certainly not my intention to advert to the work of Mr. Salmon upon "swine plague" again, but he has seen fit to assert that I have been guilty of "unfounded statements," and as we are both public servants, it is necessary that the public have still fur-

ther evidence as to which of us is the real author of "unfounded statements," and which one of us is the one in whose statements they have a right to place confidence.

It is a somewhat notorious fact that while I have been before the public, as a writer, for some twenty years, that no one has yet been able to prove me guilty of making an "unfounded statement," and I will show that the accusation is without ground this time.

In the first place Mr. Salmon simply makes an accusation without offering one single word or quotation from my writings in proof of the same.

In the next place I beg to call attention to the fact that every criticism which I have made upon Salmon's work has been made in the interests of honest scientific work and the stock raisers of this country, without any other motive than that they should know the exact facts with regard to work, which no one but a worker in the same field of research could possibly give.

For some eight years Salmon has had the chief monopoly of the opportunities to do work in original research upon animal disease in this country, and has felt perfectly safe in publishing what he pleased regarding the same.

The "unfounded statements" to which he refers in his communication to the *Breeders' Gazette* were, unfortunately for him, taken from his own published statements in the reports of the Department of Agriculture and Bureau of Animal Industry from 1880 to date. The year and number of the page has been given in each case, and the passages quoted most particularly marked as such. It is, therefore, possible for every one desiring to do so, to verify them and then to judge whether my conclusions have been "unfounded statements" or not.

The careful reader of the communication in the *Breeders' Gazette* will become at once convinced of one thing, and that is the tenor of uncertainty which runs through the whole article and which characterizes all of Salmon's work. He is never positive. It is either "I think," or "I believe," "probably," or "might," everywhere, and never the positive assertion of an exact experimentalist and logical thinker.

The communication in the *Breeders' Gazette* is headed, "Two different germs found in hog cholera." Now, if Mr. Salmon wrote that heading, he has again made an "unfounded statement;" for if it has

relation to his own work, he should have written, "Three different germs found in hog cholera;" for, in the article in question, as will be shown, he gives a description which does not correspond to any micro-organism heretofore described by him in any of his reports.

The reader of Salmon's latest communication will be kind enough to notice that, even this time, the chief of the Bureau of Animal Industry is most careful to ignore all his reports and work which was done prior to the year 1884.

Readers will see in this connection that Salmon is guilty of making a thoroughly "unfounded statement" when he says that my remarks were "based upon alleged [most terribly positive. B.] discrepancies in the published results of our microscopical work in connection with hog cholera for the years 1884 and 1885." As if there were those only!

In the first place I desire to call attention to the "unfounded" simplicity with which Salmon entirely overlooks his "discrepancies" between his work of 1880-4 and that of 1885, as shown up by me. Those were his, not "our" discrepancies! He had no unfortunate Dr. Smith to fall back on for them, and so forgets to apologize for himself. By thus ignoring them, he silently admits that they were not "un" but "well-founded statements;" and hence as he has not, and cannot, prove them to have been, or be, erroneous, and those which he alludes to of 1885 are of the same nature, I emphatically declare that, all Salmon's assertions with regard to the specific microscopical appearances of any true and specific pathogenic micro-organism in connection with hog cholera to be one continued series of "unfounded statements" down to the issue of his report of 1886.

If we did not really have Koch's (ovoid) bacteria at that time, we did have round, oblong, or oval micrococci. A change in classification does not involve any change in the description of what one has actually seen, except in the case of Salmon.

In the article in the *Gazette* Salmon notifies the public, for the first time, that the germ found by him is the same which has been found by me in Nebraska.

Many of my friends will bear me out in the statement that this is the very turn in affairs which I predicted.

Salmon endeavors to claim the priority of the discovery of this germ, which I do not care an iota about, were it the true fact, and not an "unfounded statement."

It matters nothing to the hog raisers of the United States whether this germ was discovered by me on July 7, 1886, or by Salmon on July 1, or even in 1884. All they want is that hog cholera be prevented, and we can promise them that the experiments here give the strongest warrants in that direction. If Salmon, or any one else, can anticipate us in this laudable endeavor, or discover a better method, I shall be only too glad, for it is for the country alone that I am working.

When, however, Salmon says that "I am glad to see that Dr. Billings has confirmed my work of 1884, by stating that he has found, in Nebraska, a germ which exactly corresponds, microscopically, to that discovered by Schütz, of Germany," he knows, or should know, if he remembers what he himself has stated, that he wrote on an "unfounded statement" when he said so.

He accuses me of making "unfounded statements," when any unbiased person can easily see that the accusation must necessarily fall upon his own shoulders.

From Salmon's own report of his work done in 1884 (which was published under date of 1885, and presented to the congress in the spring of 1886, and of which I only got a copy late in the summer, after numerous requests) I quoted, in the articles he refers to, his description of his "new microbe," which is as follows :

"When stained, for from one to two minutes in an aqueous solution of methyl-violet, and examined with a Zeiss one-eighteenth homogenous, they appear as elongated ovals, chiefly in pairs.

"The great number [why not all?] present a center paler than the periphery. This may be due to a greater staining capacity of the peripheral portion.

"The darker portion is not localized at the two extremities, as in the bacteria of septicæmia in rabbits, but is of uniform width around the entire circumference of the oval." (Second annual report of the Bureau of Animal Industry, 1885, page 212.)

The above is what Salmon claims to have seen and described in his report of 1885, and which he has the audacity to claim has been "confirmed" by me, here in Nebraska.

Now let us see what I said, and if it does not confirm the fact that some one else is the author of "unfounded statements" than myself!

Let us see if we cannot offer evidence which is all "that could reasonably be required to decide a scientific question of this kind?"

I said that :

“This germ according to Koch’s classification, is a bacterium.

“It is ovoid; its polar portions (or extremities) being differentiated from the middle of its body by staining quite intensely; while the intermediate portion does not take up any color, when the application of the coloring material has not been too intense.”

Now, I ask any fair-minded person, if the above description corresponds to the object described by Salmon above, or confirms his statement of his new microbe with which he worked in 1884, of which he said:

“The darker portion is not localized at the two extremities, but is of uniform width around the entire circumference of the oval.”

Does it not rather entirely contradict Salmon’s assertions of his observations in 1885, or far better, describe another organism? Does it not positively assert that the polar portions or extremities are stained; which he says, was not the case with regard to his “new microbe” of 1885, and quotes, in evidence of his assertion, its differentiation in this regard, from “the bacteria of septicæmia in rabbits,” which he negatively asserts is stained in both poles, in order to show that his “new microbe” of 1885 does not stain in such a manner, but is only marked by a fine line of staining of uniform width around the entire circumference of the oval?

In other words, he wishes us to understand that the body of his “new microbe” did not stain at all.

My readers must constantly bear in mind that it is the discovery of this “microbe” of 1884, which Salmon claims my discovery (of 1886) has “confirmed.” In the *Breeders’ Gazette* of November 11, 1886, Salmon says:

“There is an error [only one?] in the report of 1885, p. 186, where the statement is made that this germ liquefies gelatine rapidly.”

He next tells us that “this germ of 1884 was lost for a long time,” and the place taken by his old but neglected friend, a “micrococcus.”

Next he says:

“This microbe which I then called a micrococcus.”(!!!)

He did no such a thing!

The above is an example of one of his “unfounded statements.”

He did say (Report of work done in 1884, second annual report of the Bureau of Animal Industry, page 186):

"Anticipating somewhat the conclusions which we arrived at later, concerning this puzzling disease, we must say at this point: That we no longer consider a micrococcus to be the cause of all outbreaks of the disease known as swine plague."

He did say (page 229 of the same report) that "this microbe probably (!?) belongs to the genus bacterium" which is not the coccus species by any means.

But to return to the *Gazette* article.

The lost child was found again!

Salmon says "it was not encountered again in the outbreak of swine disease until the present summer"—1886.

He tells us how!

"Several months ago (in 1886) we (Salmon, etc.) obtained from an animal supposed [why not known?] to be affected with hog cholera, but which had peculiar lesions of the lungs [what were they?] a figure eight organism. This germ we have since found in other outbreaks of swine disease. * * The microscopical characters of this germ, so far as we have studied it, are identical with those of the fowl cholera microbe, and I believe (! ! !) it to be the same germ which I described in my report of 1884."

Why "I believe?"

Now, hold!

Salmon has described in his report of 1884 a micrococcus or diplococcus. (See Fig. 2, Plate I.)

When it "was encountered again," however, he says of it:

It is what has been called a figure eight organism, which stained at its two extremities, and appears to be identical with the microbe of rabbit septicaemia." (!!)

As to the identity between the objects described by Salmon as a figure eight, illustrated in his report of 1884, or that in the report of 1885, with that seen and described by myself, there is not the slightest resemblance in the coloring or description, so that knocks the bottom out of the identical priority question, if nothing else does.

A figure eight is constricted in the middle, while the one pictured by Salmon (in 1885) is an elongated oval, the ends being more blunt than rounded (see report); there is no coloring at either extremity; the internal space is clear, corresponding to his description.

That seen by me is an elongated oval with round ends, the two

ends coloring dark blue in an appropriate color; the center remains uncolored; under certain treatment the two colored ends are connected by a delicate line of colored material, leaving a clear center. If the reader will take a white bean of an elongated oval shape and paint both ends and a portion of the sides of outer cuticle blue, and leave the center of the body untouched, he will have as clear an idea as is possible to give of this organism. There is absolutely no constriction of the center, as in a figure eight, in the fully developed organism.

Now, let us see how long ago those "several months ago" were, which he tries to place back in 1884, by asserting that the *lost* germ was found again.

He is a very poor hand at covering his tracks! In the *American Veterinary Review* of August last (1886), is the conclusion of several articles by Salmon, which are entitled, "Why Pasteur's Vaccine Fails to Prevent Hog Cholera." He there refers to the report of the Imperial Board of Health of Germany, in which is the original communication of Prof. Schütz upon the bacterium of the German "Schweineseuche." This report was in my hands December, 1885, though I do not know when Salmon received it.

In the article in question, Salmon says:

"We commend that (the above) to the careful consideration of the gentlemen who have felt so certain that we were wrong in making a distinction between 'rouget' and hog cholera.

"And we tender the gratuitous observation that it would still be premature to jump to the conclusion that even the German schweineseuche is identical with our hog cholera."

It is easy to be seen then, that so late as August, 1886, Salmon did not have any idea of claiming anything "identical" between his micrococcus of 1884 and the germ first described by Schütz, and for which I was the first to claim apparent microscopical identity for one found in the swine disease in this state.

It is also easily to be seen that had not Salmon felt his position in danger; had he not really known his scientific structure was fast crumbling to pieces; that we would not have heard anything from him about the work done at the State University of Nebraska.

For ten long years he has been at work—at what?

Let Salmon show! Let him show what he has done that has been an iota of value either to his country, his profession, or science.

Salmon, when so desirous to claim that I have "confirmed" the identity of his micrococcus of 1884, seems to entirely forget that he was very careful not to claim any identity between that same germ of 1884 and Schütz', only August last—1886.

Before proceeding to the discussion of Salmon's latest report upon the American swine plague, it becomes necessary to draw attention to one fact, viz.: That up to 1886, we have direct evidence that Mr. Salmon knew of but one porcine pest afflicting the swine of the United States, and that that disease has been continually spoken of as "swine plague" by him, although in his first report he also used the popular term "hog cholera."

In the report of his investigations of swine plague in 1885, the annual announcement of which bears the date of 1886, but which was issued in the summer of 1887, we are informed that Mr. Salmon has been mistaken during these ten years of employment by our government, and that instead of one we have to bear the burden of two swine plagues, but we will let him speak for himself.

COMPARATIVE RÉSUMÉ OF MR. SALMON'S VIEWS UPON THE SWINE PLAGUE.

SALMON'S VIEWS ON SWINE PLAGUE FROM 1878 TO 1886.

1878. "Considering all the facts, there can be no doubt that these animals all died of a general disease, a disease not caused by changes in any single organ; but, on the contrary, a disease which caused all the various organic changes observed." p. 435.

1880-81. "By the investigation carried out in 1878 many important points and long contested questions respecting this disease may be regarded as definitely settled.

"Among the more important of these, I particularize:

"1. The great epizootics among swine in the West and South are the result of one and the same disease.

SALMON'S VIEWS ON SWINE PLAGUE, 1886, 1887, 1888.

"In view of the results of investigations which have shown the existence of two distinct diseases in swine, perhaps of equal virulence and distribution, a change in the nomenclature becomes necessary in order to avoid any confusion in the future. Since these two diseases have been considered as one in the past, and the names swine plague and hog cholera have been applied indiscriminately, we prefer to retain both names with a more restricted meaning.

"Using the name hog cholera for the disease described in the last report as swine plague, which is produced by a motile bacterium.

"2. The symptoms and more apparent lesions of this are definitely settled." p. 13.

1883. "This disease (swine plague or hog cholera) is still very prevalent. We know our enemy, but have not conquered him. (!) The mortality from this pest continues unabated." p. 56.

1884. "In former reports I have given the details of experiments, which, if correctly stated, demonstrate beyond question that the microbe of swine plague is a micrococcus." p. 221.

"Having obtained such results
* * * I cannot but conclude that swine plague is due to a micrococcus." p. 225.

1885. "Swine plague is caused by a specific microbe multiplying in the body of the diseased animal." p. 229.

"And applying the name swine plague to the other disease (only lately discovered) the chief seat of which is in the lungs.

"This change is the more desirable since recent investigations have shown that the latter disease exists in Germany, where it is called swine plague." p. 603.

To make my own position plain I must say :

1. That I unequivocally deny that there are two distinct diseases of swine which have been known heretofore as one disease.

2. I deny that there are two distinct germs causing two distinct diseases known by either of these names.

3. I positively assert that Salmon's assertion of a distinct germ for the disease which he now calls hog cholera is erroneous, and that the description of that object is a forgery; that it does not exist or occur in any form of the American swine plague, and that neither Salmon nor anyone else can demonstrate the presence of that object in the tissues or blood of any hog that has died of swine plague in any part of this country, if the examination is made before cadaveric changes have taken place.

4. That the object described by Salmon as the germ of the hog cholera cannot be cultivated from the tissues of any animal that has died of hog cholera or swine plague.

5. That up to the time of the issue of the report of the department, for 1885, Salmon never knew of but one disease to be called swine

plague—that up to that time he considered the micro-organismal cause of that disease to be a micrococcus, and that only—as may be seen by reading the following passage from the report of 1885: “Anticipating somewhat the conclusion which we arrived at later concerning the real cause of this puzzling disease, we must say, at this point, that we no longer consider a micrococcus as the cause of all outbreaks of the disease known as swine plague,” p. 186—and this assertion he reiterated as late as November, 1886, and yet he claims to have discovered the true germ of swine plague in “July, 1886,”—(Am. Vet. Review, April 1888)—but that is an after-thought, as the reader will easily convince himself. The interesting question now comes to mind, what has become of those outbreaks of swine plague that are caused by that micrococcus? They were not mentioned in the report of 1885, and no allusion is made to them in that of 1886, notwithstanding the fact that Salmon spent all his time from 1878 to 1885 in discovering that micrococcus and defending its position as the cause of American swine plague.

All that time was wasted. Thousands of dollars were wasted in doing—nothing! When then did Salmon discover that there were two different diseases caused by two different germs that were heretofore known as hog cholera or swine plague?

After German investigations had shown him that a germ having apparently the same appearance as that described by Detmers as far back as 1880 was the cause of a German swine plague.

Why then did he not accept the Germans’ description of that germ?

Why did he manufacture a description of an object which he cannot, nor anyone else, derive from the tissues of the swine that have died from the American swine plague?

The answer is easy! To pose as the original and only investigator on animal disease in this country!

Salmon’s description is not that of a germ at all, it is that of a spore, as everyone who knows anything of bacteriology must admit, who reads the description and studies his plates.

Salmon denies that the bacteria of the American swine plague develops spores, so that he knocks the bottom out of any argument in that direction for the present. See page 611, Report 1886, where he says:

"All the facts brought out by the study of this bacterium lead to the conclusion that a distinct spore state does not appear either within the animal body or in nature."

The writer must endorse that assertion at present in the face of some very positive natural facts which strongly point to the probability that a permanent spore may be developed by this germ under certain unknown conditions.

According to Salmon, then, we have two distinct porcine diseases in this country, both having approximately "Equal violence and distribution."

He gives two reasons for this assertion :

1. That they are caused by two distinct germs.
2. That the pathological lesions are different; so different that one can easily distinguish one disease from the other.

The first or hog cholera is an enteritis.

The second is "pneumonia," or as he speaks of it, "a chronic pneumonia."

Why Salmon should designate this disease as a "chronic pneumonia," is something beyond comprehension unless it was an effort to appear original, as Schütz had defined the German disease as an "acute, infectious pneumonia."

A disease that generally kills in ten to twenty days cannot be called "chronic" by any means, though there may be many cases in which the pneumonia may become chronic and the animal finally die of marasmus or general emaciation, but there are other things playing a role in the performance than the inflammation of the lungs.

We must now return to Salmon's first condition for the differentiation of hog cholera from swine plague, viz.:

The germs are different. For the description of his germ of hog cholera he refers us to his report of 1885, where he says:

"When stained for from one to two minutes in an aqueous solution of methyl-violet, they (the germs) appear as elongated ovals, chiefly in pairs. The greater number present a center paler than the periphery. The darker portion is not located at the two poles," etc. p. 212.

I desire to call attention to the fact that Salmon admits that some do not present a center paler than the periphery, and also that he is positive in asserting that the darker portion is not located at the two poles.

It has been previously said that the above is a description of a spore, and not a germ, and Salmon's illustrations in both the reports of '85 and '86 will bear out that assertion.

Of this nonentity of Salmon's, the writer said:

"If Salmon knows anything of the chemical affinities of bacilli, cocci, and bacteria, except spores, he knows that the description which he has given of this new microbe does not apply to any known form of bacteria, but to spores."

He seems to have then felt that he was treading upon dangerous ground in issuing such a description, for on page 196 (1885) he says:

"The pale center was very distinct, suggesting very strongly of spores."

To which the writer added: "What, then, is the distinguishing characteristic of spores"?

Hueppe, one of the most able authorities, gives us the generally received definition as follows:

"That by the employment of aqueous or diluted alcoholic solutions the spores do not color."

Their outside cuticle does, however, and that is just the object which Salmon has described. But the bacteria of hog cholera do not develop spores!

Salmon was just as sure there was but one hog cholera in the country and that that one form was caused by a micrococcus up to 1885 as he was that that same hog cholera or "swine plague" was caused by a specific microbe, and that "this microbe belongs to the species bacterium," in '85, as he now is that there are two causes of swine plague and two diseases.

What dependence can be placed on such a contradictory observer?

The records of scientific investigation can be searched in vain for such a mass of contradictions as appear in Salmon's publications.

We must again call attention to Salmon's description of the manufactured germ of 1885:

"The darker portion is not located at the two poles as in the bacterium of septicæmia in rabbits." p. 212.

In the report of 1886 he says:

"In most forms there is a slightly thicker border at the ends than at the sides of the short, rod-like bodies," p. 610. (See Fig. 2, Plate I.)

Here is one concession !

The ends do stain more than the sides !

This time we have no qualification about the ends staining somewhat. It is not "the greater number," which "present a center paler than the periphery," but "in most forms there is a slightly thicker border at the ends than the sides."

The reader will be kind enough to remember that Salmon has asserted that there are two distinct swine plagues in this country caused by two distinct germs. I will now give evidence that he is not sure on this question, as well as show further contradictions of a most disgusting character.

Early in March, 1886, he sent a Dr. Rose to Nebraska to make observations as to what was going on here, but ostensibly to collect material for study in Washington. It seems pretty expensive work to send a man that distance to gather specimens from ten hogs, when some one on the ground could have done it equally well in the manner it was done. Had Mr. Rose been supplied with cultivating tubes and a spirit lamp, and even instructed how to use them, he might have done himself some credit. As it was, "in only one case was the result successful." Page 623, 1886.

Of these results Salmon says :

"This new microbe, identical morphologically with the bacterium of hog cholera already described."—*Ibid.*

How then could it be a new microbe ?

Again he says :

"The disease caused by this germ, in its duration, symptoms, and lesions in rabbits and mice, cannot be distinguished from that caused by the bacterium of hog cholera." p. 627, 1886.

Again that

"These lesions (in hogs this time) were as intense as any produced by feeding hog cholera bacteria obtained in the East," and the "identity of the two bacteria from Nebraska and the East was thus completely established." *Ibid.*

The above assertion is positive. The diseases are identical according to Salmon. On the same page, however, Salmon is not so positive about this, for he says :

"A liquid culture of the blood seemed a pure culture of a motile oval bacterium resembling closely the bacterium of hog cholera." (!)

On the next page, 628, 1886, Salmon proceeds to give the "differential characters of the hog cholera bacterium from Nebraska," of which he had said on the previous page that "the identity of the two bacteria from Nebraska and the East was thus completely established."

How then could there possibly be any "differential character" on the part of the Nebraska germ?

It will be interesting to learn what these "differential characters of the hog cholera bacterium from Nebraska" were:

"The bacterium, when stained on cover-glass preparations from the spleen and other viscera, closely resembles the one found in the disease prevalent in the East, so that it is impossible to distinguish them in this way. Both stain well in an aqueous solution of methyl-violet in from two to five minutes, and show a well-stained narrow periphery around a pale center. This may be due to the presence of a dense envelope obstructing the inward movement of the coloring matter.

"A few minor differences revealed in the various culture media indicated that the two microbes were not alike in every way.

"The first difference was observed in liquid cultures. Within twenty-four hours after inoculation from the spleen or blood the culture liquid became turbid, and upon its surface *a complete membrane* was present in nearly every case. This whitish membrane is not homogeneous, but *made up of patches* of varying thickness, and when shaken, slowly settles to the bottom in lumps and floeculi. The microbe of Eastern outbreaks does not form a membrane within several days after inoculation, and then only when the tube remains perfectly quiet. It appears as a whitish ring attached to the glass, and is rarely found covering the entire surface. When a number of successive inoculations are made a week apart the later cultures are quite apt to form membranes after a few days' standing. Thus one microbe forms a membrane very speedily; the other only occasionally, and then quite tardily.

"In these liquid cultures both exhibit, during three or four days after inoculation, very active spontaneous movements. Sometimes masses of five to ten bacteria may be seen moving actively to and fro and at the same time revolving about themselves.

"In the same culture liquid the microbe from Nebraska seemed to grow more vigorously, so that at the end of two or three days the liquid became turbid, and the deposit in the bottom of the tube was very abundant after one or two weeks.

"Both fail to liquefy gelatine. A very slight but significant difference was observed in this medium also. It was found that when lin-cultures were made on plates of gelatine, in order to test the purity of

liquid cultures, *the microbe from Nebraska failed to develop, while the microbe from the East invariably grew as described in the preceding report.* This observation was made so uniformly with every culture that it *became later a means of distinguishing* the two forms when the cause of this behavior became known. Such lines, after a few days, appeared as an aggregation of mere points under a 1-inch objective, and did not enlarge, or else there was no indication of any growth whatever.

"In gelatine the bacterium from Nebraska grew very well both on plates and in tubes, and the bacterium from the East grew much better than in the previous preparation of gelatine, thus showing that an alkaline medium is best for the bacterium of hog cholera, and that the Nebraska variety is by far the more sensitive, and fails to multiply unless the reaction is fairly alkaline. On gelatine plates the colonies are somewhat darker and more coarsely granular when viewed by transmitted light than those which develop from the Eastern variety.

"On the surface of beef-infusion peptone agar-agar, made slightly alkaline with potassium carbonate, both bacteria grow very vigorously when kept in the incubator at 95° to 100° F. On potato both grow as a dirty straw-colored layer at the ordinary temperature, so as not to be distinguishable."

This is contradicted as to the hog cholera germs by Salmon's remarks in his report of 1885, where he says :

"On boiled potato the bacterium grows very well. It seems to be a far better substratum than beef infusion peptone gelatine. The bacterium manifests growth by first staining the white cut surface of the potato at the place of inoculation with a chocolate color, gradually turning quite dark and spreading over the entire surface. In the latter stage it resembles the discoloration frequently observed on boiled potatoes standing for a day or two. The growth itself begins in the form of small round masses which gradually unite into a patch $\frac{1}{2}$ mm to 1 mm thick. This patch spreads slowly by lateral extension and its straw-colored, slightly greenish surface contrasts strongly with the dark, bluish-red background of the potato. This description applies to growth at a temperature of 65° to 80° F. In the incubator, at 95° F., the multiplication was more rapid and abundant." p. 215.

"From the comparative plate cultures and from potato cultures, both bacteria, when inoculated into liquid media, showed the characteristic difference already mentioned. On the following day one culture would be covered with a membrane, the other not. In milk both multiply without producing any microscopic change." pp. 628-9—1886.

With such essential points of differentiation between two micro-organisms, how could Salmon have said that "the identity of the two bacteria from Nebraska (1886) and the East (swine plague 1885; hog cholera 1886) are thus completely established"?

SALMON'S NEW SWINE PLAGUE GERM.

In the letter to the *Breeders' Gazette*, Salmon wrote :

"I am glad to see that Dr. Billings has confirmed my work of 1884 by stating that he has discovered a germ which exactly corresponds in its microscopical appearances to that discovered and described by Schütz in the *Schweineseuche* of Germany."

The germ described by Salmon in his report of 1885 in no way corresponds to that discovered and described by Detmers, Schütz, or myself.

Above he has said that Dr. Billings has confirmed his work (not report) of 1884.

In this report he seems to have forgotten all about that observation. In this report he says :

"Although the investigations concerning the nature of this microbe are scarcely begun," etc. p. 659, 1886.

How then could he have described it in 1884 ?

On page 618 he admits that somebody has found the bacterium of his swine plague before him, but in a very peculiar manner for an honest investigator. He says :

"In view of the fact that another bacterium [he is writing about his h. c. humming at the time] has been recently found associated with lung disease and is probably the cause."

If, then, Salmon's investigations "are scarcely begun," and as it is the germ discovered by Dr. Billings alluded to above of which he speaks, how in the name of ordinary intelligence could my work confirm that of Salmon in 1884, the very trustworthiness of which he now shatters all to pieces? How then could he have said he discovered it in July, 1886 ?

Salmon's study of this germ can certainly be "scarcely begun," if we are to judge from his very meagre description, but this much we will quote :

"The two extremities of the longer axis are deeply stained. Between these colored masses a transverse band remains transparent without any color." p. 671, 1886. See Plate 1, Fig. 5.

I have already given the following short description of the appearances of this organism :

"This germ, according to Koeh's definition, is a bacterium. It is ovoid, its polar portion being differentiated from that in the middle of its body by staining quite intensely, while the intermediate portion does not take up any color when the application of the coloring material has not been too intense. It colors best in methyl-violet, gentian-violet and methylen-blue, in the order named; also well in methylen-green, but not so well in fuchsine, and not at all in dahlia or negrosin: *i. e.*, micro-morphologically it bears a marked resemblance to the organism described by Loeffler and Schütz, of Berlin, as the cause of the German 'Schweineseuche'—swine plague."

Let us see what Salmon says in another place, where we find a description of this new germ :

"One grew in both tubes, which was more carefully examined, because it resembled the bacterium of hog cholera very closely. When stained, however, each individual is resolved into a pair of ovals, or very short rods with rounded extremities. A deeply stained narrow border surrounds a comparatively pale body."

Of the other germ, Salmon wrote the same.

"The darker portion is not localized at the two extremities, but is of uniform width around the entire circumference of the oval." Report 1885, page 312. See Figs. 3 and 4, Plate 1.

Salmon seems to have remembered this contradiction, so he corrected himself at once as follows :

"There seems to be slightly more stained material at the two extremities than in the bacterium described in the last report." Page 661, 1886. See Figs. 3 and 4, Plate 1.

The reader will observe that Salmon is not sure about this. It "seems to be" so, but a glance at his illustrations will show something more than a "seeming" difference in this direction, and above he has told us that "the two extremities of the longer axis are deeply stained." This is not "seems to be, or slightly."

The original discovery of the germ of swine plague does not belong to me, however, but to Dr. Detmers of the agricultural college of Ohio, and was made in 1879, who gave a very fair description of the object, though mistaken with regard to the vital phenomena.

SALMON'S CONTRADICTIONS WITH REGARD TO THIS GERM.

The first description of it is in connection with eight post-mortems made in Illinois, and described on pages 660-661, of which he says :

"Besides the cultures mentioned in the autopsy notes at least ten others were made at the time. * * None of these showed any signs of growth."

What shall we say then when a few lines further down the page we read :

"We will now proceed to a description of the bacteriological investigations of cultures, 'none of (which) showed any signs of growth,' yet in two tubes inoculated from No. 6, two microbes were found which deserve attention." (!)

The next line is interesting.

"One (germ) grew in both tubes which was more carefully examined because it resembled the bacterium of hog cholera very closely." (!)

Salmon will now tell us how "closely."

In his summary he tells us that the "bacterium of H. C." is "motile in liquids" while that of swine plague is "non-motile in liquids,"—p. 674, Ibid. "No spontaneous movement can be observed,"—p. 672 ; see also p. 682, where the same differentiation is pointed out.

The fact is, the true germ of the American swine plague, the one described by Detmers and the writer, has a modified motion in fluid cultures, and Salmon knows it and has recorded it, as may be seen in the following quotations, although they directly contradict his previously quoted assertions. On page 661 he says of the germ which he says is "non-motile in liquids," that "in liquids it is actively motile." On page 662 he again says: "In one of the tubes just described a motile bacterium * * * " which has reference to the same germ. Then why did Salmon say that it was "non-motile"? The reason is easy to discover.

Because Schütz had said the German organism of "Schweineseuche" had no independent movement in blood drops, which is not saying that it does not move, and Salmon this time falls back on Schütz for support, and has therefore proclaimed the two diseases identical.

FURTHER EVIDENCE.

In his summary showing the difference between his manufactured germ and the true germ of the swine plague, Salmon says that the latter "fails to grow on potatoes" (p. 674), in order to distinguish it from the former, which grows luxuriantly on potatoes, but he apparently forgets that on page 661 of the same report (1886) he had already written of this same germ of which "the growth on potatoes fails," that on "potatoes a thick straw colored shining layer of nearly smooth surface forms, which grows very vigorously and gradually covers the entire surface of the potato."

This is correct, and his illustration of such a growth, which, however, he ascribes to his imaginary germ, exactly corresponds to that observed by me for the true germ, but I have used the term "coffee colored" to describe it, which is the color he gives in his illustration, and not "straw-colored."

In this regard it may not be uninteresting to note that Loeffler says that the germ discovered by him does not grow on potatoes, although he made a number of attempts, and that Schütz claims that that germ is identical with the one which he has shown to be the cause of the German swine plague, but not entirely with good reason. It is singular that Schütz does not mention a thing about the growth of his germ on potatoes. The germ of the German swine plague does grow on potatoes, and so does that of the French. (See proof later.)

Attention has already been called to Salmon's saying that his germ of swine plague fails to grow on potatoes, while that of his hog cholera does grow on them. Hence I desire to call attention to another unwarranted statement, which follows on the last quotation from him, where he says the germ of swine plague does grow on potatoes, and then tells how this growth differs from that of his hog cholera non-entity in the following words:

"This growth is brighter in color and more abundant than appears in the potato cultures of the bacterium of hog cholera." Ibid., 1886, p. 661.

That should settle the potato question.

Why did not Salmon show this differentiation in his plates?

OTHER CONTRADICTIONS BY SALMON.

With regard to this bacterium of swine plague, Salmon again says

"That it was not the bacterium of hog cholera was shown by an utter want of pathogenic properties when inoculated into mice and rabbits."—*Ibid.*, 1886, p. 662.

On page 663 of the same report he says, of the same object:

"This microbe was therefore fatal to mice, rabbits, and pigs.

"Rabbits, mice, and pigeons were thus shown to be susceptible." *Ibid.*, 1886, p. 665.

"Mice destroyed, but not invariably, in two to six days." Summary, p. 674, 1886.

"In rabbits inoculation destroys life in from three to six days." *Ibid.*"

There is more evidence of this kind, but it is unnecessary to quote it.

I must also deny another assertion of Salmon's, which is, that the true germ of the American swine plague, or his pneumonia germ, causes sclerosis of the liver. It does not.

The correctness of my assertion is well shown by the following quotation, which shows his utter want of any knowledge of the principles of pathology. He says:

"There was, moreover, a partial sclerosis of the liver in most of the animals examined, which we never encountered in hog cholera.

"We must remember, however, that of these eight cases five were killed, perhaps in the earlier stages of the disease, before the lesions were well marked." p. 661, 1886.

I will only say that any one who knows anything about interstitial inflammation of the liver knows that it is absolutely impossible for it to occur and be caused by bacteria in the early stages of a disease of not over twenty days' duration, even in protracted cases.

Salmon never saw one single case of sclerosis hepatitis due to any bacterium connected with the American swine plague!

The terrible looseness and inaccuracy which characterizes this report of our "Chief of the Bureau of Animal Industry" has been pretty well illustrated, but to complete the picture the following quotation is appended:

"The failure to produce the disease in even a small proportion of animals by the injection of liquid cultures raised the question whether the cultivation in itself did not attenuate the bacteria. (?) Consequently

two experiments were made by inoculating with blood directly. Numerous gelatine cultures of heart's blood had demonstrated the very small number of bacteria compared with the number present in the spleen.

"September 10.—A pig dying with the disease was killed, the heart carefully exposed, and the blood drawn with a disinfected hypodermic syringe. Nos. 329 and 333 received subcutaneously 5^{cc} each, one-half in each thigh. No. 329 in a few days lost its appetite; became weak and stupid. Found dead October 5; slight local swelling at the points of inoculation; superficial inguinals greatly enlarged; hypostatic congestion of lungs; complete necrosis of mucous membrane in cæcum; large, scattered ulcers in colon, showing as whitish patches on serous surface, and encircled by a crown of enlarged blood-vessels; bacteria in spleen.

"No. 333 slightly ill for a time; fully recovered. Died December 2 with no other lesions than engorgement of liver. No signs of former ulceration." pp. 616–17, 1886.

The above certainly "raises" the question how a "fully recovered pig" could die so suddenly; and again, how "signs of former ulceration" could be seen in a "fully recovered pig," that was but "slightly ill for a time." The careful reader of the preceding quotations must now be ready to admit the following facts:

1. That, according to Mr. Salmon, the cause of swine plague from 1880 to 1885 was a "micrococcus," the evidence furnished being all that could be required to decide a scientific question of this kind.

2. That, according to the same authority, there was still but one swine plague in this country in 1885, the cause of which being his "new microbe," as he "no longer considered a micrococcus to be the cause of all outbreaks" of that disease.

3. That, according to the last report of the Chief of the Bureau of Animal Industry, there should be three swine plagues in this country, as he has described in that report three microbes, which have differential characteristics which distinguish them from each other, viz.:

- a. The swine-plague-hog-cholera article of 1885–86.

- b. Mr. Rose's peculiarity from Nebraska.

- c. The true germ, which, it will be shown, these people have never demonstrated as existing. As the reader may be inclined to doubt the above, we commend him to the following quotations:

Salmon says:

"The appearances and morphological differences of the microbes

discovered in hog cholera and in the infectious pneumonia which we now call swine plague, are illustrated by the photo-micrographs reproduced in Plates VII., VIII., and IX. 1. The microbe of hog cholera is seen in Plate VII., Fig. 1, as it appears in the tissues of the body. Most of the germs in this case are sufficiently elongated to be classed as bacilli. In cultures where more rapid multiplication occurs the microbe is shorter and assumes an oval form (Plate VII., Fig. 2). 2. The microbe from the Nebraska outbreak of hog cholera, referred to above, maintained a distinctly rod-like form, however, even in liquid cultures (Plate VIII., Fig. 1). 3. The micrococcus of swine plague, which is plainly oval in the tissues and in the blood, as shown in Plate IX., Figs. 1, 2, becomes very nearly or quite spherical in liquid cultures (Plate VIII., Fig. 2). [False as false can be. See Salmon's illustration.] This is also very nearly the form seen in gelatine cultures." p. 674.

As the master is, so must the assistant be. We find this melancholy farce is endorsed by Dr. Theobald Smith, as follows:

"A course of lectures has just been given at Cornell University on 'Pathogenic Bacteria and their Relation to Hygiene,' by Theobald Smith, M.D., of the class of 1881, Chief Assistant in the United States Bureau of Animal Industry. The lectures were largely attended by the faculty and students, and were supplemented by a paper before the Agricultural and Natural History Societies on the three diseases which are commonly confounded under the term 'swine plague,' but which Dr. Smith has shown to be caused by three distinct germs."—*Rural New Yorker*, June, 1887.

FURTHER EVIDENCE OF MR. SALMON'S UNRELIABILITY.

Having conclusively shown that no such organism as Mr. Salmon's "swine-plague-(1885) hog-cholera" psychosis exists in the porcine pest of America, and as conclusively demonstrated that but one porcine pest exists in this country, it now remains to show, and from Mr. Salmon's writings, again, that he does not give a particle of evidence which can be relied upon "to decide a scientific question of this kind," showing that he ever experimented with or even saw the true germ of swine plague.

On the other hand he does give most conclusive evidence that he never has seen it or experimented with it.

It will be remembered that this organism is the true germ of swine plague, and that Mr. Salmon claims it is not, but that it causes a "chronic pneumonia" in a disease of less than two weeks' duration

at the extreme, when recovery does not occur. According to Mr. Salmon, "it is a disease in which the principal lesions are to be found in the lungs."

It will also be remembered that this Mr. Salmon cried very loudly at the meeting of the United States Veterinary Association, March last, for proof that the swine plague is an extra-organismal infection.

Now I ask for proof from Mr. Salmon that he has ever experimented with the true germ of swine plague, or that of his "chronic" pneumo-psychosis?

I ask him, "Where are the records which demonstrate this proposition put forth in such explicit terms"? "Have you seen them"? "I have not"!

And, what is more, I cannot find them! With his forged germ Mr. Salmon gave us records of experimental inoculations which, if ever made, go to show that both the pulmonary and intestinal lesions of swine plague were produced thereby, all of which is false as false can be, but for this "swine-plague-chronic-pneumonia microbe" he does not even give us that satisfaction. In no single case does he give evidence that he produced any pneumonia by inoculating hogs with cultures of this germ, and, what is more, in no single case does he describe any of the induced lesions common to swine plague, but he does introduce a new lesion—a specific lesion—sclerosis, or, as it is wrongly termed, "cirrhosis, hepatitis." Another "chronic" psychosis, which it is impossible to produce in any such disease as swine plague.

MR. SALMON'S EXPERIMENTAL EVIDENCE THAT HE NEVER SAW
THE GERM OF SWINE PLAGUE.

"Two pigs (Nos. 287 and 289) were inoculated September 11 from a culture of the rabbit. Each received beneath the skin of the thigh $2\frac{1}{2}$ cc of the culture liquid. No. 287 became dull and lost its appetite several days later; eyes discharging. September 28 the animal became delirious and ran blindly about the pen; dead next morning. The only observable lesions were local swellings two inches across and one-fourth to three-fourths inch thick, with centers which were beginning to soften. Blood very dark, not coagulated; a few petechiæ on epicardium. The liver was very pale, sclerosed; the medulla of kidney deeply reddened. No. 289 died September 21, after exhibiting the same symptoms; local swellings as above, without indications of softening; the connective tissue and fat of the whole body of a deep yellow color; liver very firm, bloodless, and of a peculiar yellowish

red color throughout; medulla of kidneys deeply reddened; two large cysts in the right one. In neither case was the alimentary tract diseased. In both there was cirrhosis of the liver, producing in the second animal a general jaundice. From neither were cultures of the inoculated microbe successful, though blood from the heart, the spleen, and the liver were used. The tubes remained sterile." 1886, p.p. 662-63.

"Of the two pigs inoculated (Nos. 330 and 331), No. 330, which had received the larger dose (5^{cc}), died in nine days, after exhibiting the same symptoms as those manifested by the two former cases—debility, loss of appetite, inflamed eyes. In this animal there was a similar condition of the liver, together with a deep yellow staining of the connective and adipose tissue generally. Cultures negative. No. 331 died thirty-five days after inoculation. In this animal there was a less pronounced pathological change in the liver. Icterus present. No cultures were made." p. 665.

"October 30, two pigs were inoculated with the microbe from Geneseo, and an equal number with that from Sodus, Ill. The pigs received subcutaneously each 5^{cc} of a beef-infusion peptone culture. Of the four only two died. One of these had been inoculated with the microbe from Sodus. On the third day both eyes were discharging, the animal looking unthrifty and becoming weak and thin. It died on the eighth day. In brief the lesions were as follows: Fat and connective tissue in general yellowish. Both ventricles of heart filled with large washed clots and semi-coagulated blood. Liver very firm, of a dirty red-lead color. On cutting into it a gritty sensation is transmitted to the hand. Venous stasis of the abdominal vessels. Other organs and intestinal tract normal. Cultures failed to detect the microbe in the spleen, liver, and blood.

"No. 363, inoculated with 5^{cc} from a culture of the microbe obtained from Geneseo, Ill., showed inflammation of the eyes a few days after, which disappeared in a week. At the same time the animal looked unthrifty. It had apparently recovered two weeks after inoculation, when it again became unthrifty and weak. The abdomen became enlarged and it was unable to rise. Found dead December 27, nearly two months after inoculation. At each point of inoculation on the thigh an encysted mass was found in the subcutaneous tissue as large as a marble. The contents were softening and inclosed by a fibrous wall. Lungs hypostatic. Pulmonary vessels and right heart filled with a firm clot. Liver very much contracted, especially the lobes on the right, and streaked with depressed lines and furrows. The peritoneal covering on the cephalic aspect was very much thickened, in some places uniformly, in others in a mesh-work corresponding to the interlobular tissue. The acini of this side were very small. On the caudal aspect they were in some places very large and bulging.

On section this transition from large below to small above could be easily traced. Gall bladder filled with a thick prune-juice-colored bile. Inflammatory adhesion between rectum and cæcum. The mucous membrane of the large intestine of a dull red color, probably due to a passive congestion. No ulceration anywhere to be seen. The intestine itself was very much distended with dry, half-digested feces of a yellowish hue. Four liquid cultures made from blood of heart remained clear.

"This case is interesting in that the inoculation caused a cirrhosis of the liver, which became indirectly fatal by destroying in great part the normal functions of this organ." p. 668.

"November 18, two pigs (Nos. 374 and 375) received hypodermically 5^{cc} (one-half into each thigh) of a beef-infusion peptone culture two days old, derived from a gelatine culture (rabbit) about one month old. Three pigs were placed with these to determine whether the disease was infectious. In both animals two days after inoculation the sclerotic became deeply reddened. This congestion was followed by discharge, which gummed the lids together for a part of the time. In about a week these symptoms gave way and the eyes became jaundiced. The eyes of the three check-pigs were not affected. No. 375 died November 25, one week after inoculation. The subcutaneous connective tissue of a deep yellow color. The points of inoculation occupied by cysts filled with a blood-stained serum. Blood black, partially coagulated. Hypostatic congestion of lungs. Purplish spots beneath pleura and in parenchyma; some lung worms present. Liver very pale, bloodless, very tough. The sclerosis general and the contraction of the connective tissue has made the caudal aspect very concave. Removed from the body it resembles India rubber, as it retains the same form in whatever position it is laid. Gritty sensation when cut. The gall bladder filled with semi-liquid, dark brown bile, resembling plum juice, surrounding a mass of putty-like consistency and of the same color. The papillar opening of the common duct into the duodenum contained a plug of gelatinous mucus, and when the duct was slit open it contained mucus only. The walls were not even bile-stained; the secretion of bile had ceased some time past. When the liver was cut the section was of a dirty reddish-yellow color throughout; no blood flowed from the vessels; when scraped the cellular elements of the acini came away readily, leaving the tough interlobular tissue *in situ* as a honey-combed mass.

"Sections hardened in alcohol and stained in alum-carmin showed a large amount of connective tissue as compared with the normal liver. This increase was general. In the parenchyma of the lobules there were circumscribed areas in which the protoplasm of the cells stained very feebly, while the nuclei were either shriveled or else replaced by a group of granules. The characteristic trabecular struct-

ure in these areas was more or less destroyed. Almost every lobule examined contained these altered regions, which were situated as a rule near the periphery.

"There was a very marked venous stasis of the portal circulation, characterized by an overdistension of the vessels, bringing even the smallest into view. The vasa recta of kidneys very prominent, giving the pyramids a bright red appearance. Serum in the abdominal cavity of a deep yellow. This yellow tinge is present in the fat around the base of the heart. The urine deep yellow, the mucosa of bladder stained with the same color. The urine readily gives, with Gmelin's test, the colors characteristic of the bile pigments. The intestinal tract normal throughout, save what changes arose from the general stasis of the portal circulation. The stomach empty and coated with a viscid mucus.

"The injected microbe which without doubt caused these lesions could not be found. Three tubes of culture liquid were inoculated each with three to four drops of the blood from the heart. They remained sterile. Bits of spleen and liver about $\frac{1}{2}$ cm cube were dropped into tubes. They also remained sterile." p. 669.

The above includes every particle of evidence recorded by Mr. Salmon in favor of his having obtained the true germ of swine plague, or that it causes a specific pneumonia.

That he fails thoroughly in demonstrating these facts is self-evident. To have done so he should have

1st, Found the bacterium.

2d, Have cultivated it.

3d, Have induced pneumonia by inoculating healthy swine with it.

4th, Have found it again in the tissues and blood of those swine.

Neither of these postulates have been fulfilled by Mr. Salmon, for if he had had this germ in his hands and cultivated, as he says he did, and inoculated all these pigs, he should not only have induced pneumonia in some of them, but in all have found the germ again in the tissues, for the technical work is too ridiculously easy to permit of any such negative results as those reported by this so-called investigator. As has been said, he has not done this.

What he has really done I leave to the reader to decide. It is certainly beyond my ability.

Comment unnecessary !

SUMMARY OF SALMON'S CONTRADICTIONS.

"The following comparative table sets forth briefly the differences between the bacterium of hog cholera and the microbe which has been found associated with pneumonia in pigs and described in the preceding pages :

HOG-CHOLERA BACTERIUM.	MICROBE OF PNEUMONIA.	CONTRADICTIONS AND ABSURDITIES IN THE SAME REPORT.
<i>Morphological and biological properties.</i>	<i>Morphological and biological properties.</i>	
1. Ovals varying in length from 1.2 ^{mm} to 1.8 ^{mm} .	1. Ovals varying in length from .8 ^{mm} to 1.2 ^{mm} . (In both species the size is very variable, according to the stage of growth and division and the culture medium.) This microbe is in general much smaller than the bacterium of swine plague.	Rose's Nebraska germ is forgotten ! No evidence given that it produces pneumonia in experiments.
2. Stains around periphery, with a slight increase in the width of the stained border at the extremities; observed chiefly in the tissues of animals. In cultures may stain entirely. (See original description.)	2. Stains in process of division at the two extremities only.	
3. <i>Motile</i> in liquids.	3. <i>Non-motile</i> in liquids.	"In liquids it is actively motile." p. 661.
4. Grows actively on potato.	4. Growth on potato fails.	"In one of the tubes a motile bacterium." p. 662.
5. Resists drying for one to two months.	5. Resists drying only a few days.	"On potatoes, a thick straw-colored layer of nearly smooth surface forms." p. 661.
6. Multiplies for a time in drinking water and remains alive at least four months.	6. Does not multiply in drinking water, and is entirely destroyed in a few weeks.	"The growth is brighter in color and more abundant than appears in the potato cultures of the bacterium of hog cholera." p. 661.
<i>Pathogenic effects.</i>	<i>Pathogenic effects.</i>	"That it was not the bacterium of hog cholera was shown by an utter want of pathogenic properties when inoculated into mice and rabbits." p. 662.
1. In small susceptible animals subcutaneous inoculation causes but slight local reaction.	1. Local reaction usually very severe and extensive.	"This microbe was therefore fatal to mice, rabbits, and pigs." p. 663.
2. In mice it always produces a disease lasting from eight to sixteen days; spleen enormously enlarged; liver enlarged and containing numerous foci of coagulation necrosis.	2. Mice destroyed, but not invariably, in two to six days.	"Rabbits, mice, and pigs were thus shown to be susceptible." p. 665.
3. In rabbits the disease produced by inoculation of small quantities of culture liquid into thigh lasts from six to nine days. Great enlargement of spleen; enlargement of liver and centers of coagulation necrosis.	3. Same mode of inoculation destroys life in from three to six days. Extensive local sero-sanguineous, later purulent infiltration and thickening; plastic peritonitis; spleen not enlarged.	

Local lesion: circumscribed necrosis of muscular tissue. Lungs usually have hemorrhagic foci or more extensive lobular pneumonia.

4. Same mode of inoculation destroys guinea pigs with a few exceptions. Lesions quite the same as in rabbits. May live fourteen days.

5. Pigeons destroyed by large doses. Bacteria in internal organs.

6. No fowls destroyed by inoculation.

7. Pigs are either not affected by hypodermic injection, or else a severe disease follows, characterized by hemorrhages in all organs. Bacteria present in large numbers in internal organs.

8. Feeding cultures after starving for a day produces extensive necrosis of mucous membrane of large intestine; inflammation and occasional ulceration of stomach and ileum.

4. Guinea pigs somewhat more refractory; extensive local lesions as in rabbits; occasionally plastic peritonitis; die in four to six days; spleen not enlarged.

5. Pigeons also susceptible to large doses. Bacteria absent from internal organs.

6. Large doses kill fowls. Very extensive local infiltration and destruction of muscular tissue.

7. Large doses cause acute sclerosis of liver, with icterus.

8. Feeding cultures produces no effect whatever. Page 674, 1886.

Humbug! Icterus hepatitis is only occasionally seen, but never sclerosis.

Another misstatement, as will be shown by most positive evidence.

THE SPECIFIC AND ONLY GERM OF SWINE PLAGUE.

Having, in previous remarks, shown that Mr. Salmon has not done anything in contributing to the discovery of the cause and nature of swine plague, while he has done everything possible to prevent others from investigating that, as well as other animal diseases, it now remains for us to consider what others have done in investigating this disease.

The first person to enter upon the investigation of this porcine pest was Dr. Klein, of England, and although he really failed in contributing anything of value to its etiology, still it can be easily shown that this observer did have the true organism under his eye, but failed to recognize it as an independent individuality, mistaking it for a vegetative phase, or condition, in the development of another organism—a bacillus—which he thought to be the cause. Klein's first inves-

tigations were made in 1876. We have already quoted his latest assertions upon this point, but must again do so in this connection. Under the heading, "*Bacillus of swine plague*," he says:

"In a report to the Local Government Board for 1877-78, I have shown that, in this acute infectious disease, the affected organs contain a form of bacterium in morphological respects identical with *bacillus subtilis*, *i.e.*, consisting of longer or shorter motile rods, capable of forming spores; further, that artificial cultures of these bacilli cause the disease in pigs after inoculation; and lastly that mice and rabbits become infected with this disease after inoculation with material derived from the diseased organ of the pig." *Micro-organisms and Disease*, 1885, p. 94.

Any one who reads the experimental testimony of Dr. Klein will become convinced that he did, indeed, produce swine plague in healthy pigs by inoculating them with his cultivated material; at the same time any competent person, thoroughly acquainted with the results of our own and other investigations, will at once become convinced that Dr. Klein did not produce the inoculated disease with the bacillus—"identical with *bacillus subtilis*," because no such micro-organism has any etiological connection with swine plague proper in this or any other country. Dr. Klein's success, however, is to be easily understood by referring to page 97 of the same work, where one can see two cuts—figures 63 and 64—in which he represents an entirely different organism than "*bacillus subtilis*," and shows, without question, that he mistook the objects here pictured for vegetative forms (spores) of that organism, and not for an independent species. We have taken the liberty of copying these two cuts in figures 3 and 4, plate II. of this report, as well as Klein's illustrations of *bacillus subtilis*—figures 39, 40, pages 75-6 (Plate II., Figures 1 and 2) of the same work, which will at once enable the reader to see that there is no point of resemblance between the two organisms described by Klein as identical. In Klein's figure 40 (this report, Plate II., Fig. 2) *bacillus subtilis* is represented in the condition of spore development, which in no way corresponds to any phase represented in his figures 63 and 64; in fact the germ of swine plague does not produce spores. The only point which it is necessary to call attention to in Klein's work is, therefore, that he did see and illustrate the true germ of swine plague in 1876. By comparing Figs. 3 and 4 of Plate II. with Fig. 5, the reader will see the resemblance between the objects seen by Klein

and the true germ. Klein makes no change in his description in Virchow's Archiv., Vol. 95, 1885. Just here is the proper place to again remark that we were the first to discover this micro-etiological organism in the tissues of an English hog that had died of swine plague. The discovery came about in this way. In the fall of 1886, there worked in my laboratory a Dr. Thomas Bowhill, M.R.C.V.S., and a recent graduate of the "New Veterinary College," Edinburg, Scotland. Dr. B. had, unusually for a veterinarian, given some attention to microscopy, in the laboratory of Professor Hunter, while pursuing his veterinary studies, and thus came into possession of quite a fine selection of slides representing the lesions in a number of animal diseases. Among these were some specimens of the tissues of swine plague lesions, stained in the ordinary way. These were carefully dismounted; the gum dissolved and washed out. They were then restained with methyl-blue, and the same organism, to be later described, found in them. This discovery, in connection with the description of the disease by competent British observers, completely established the fact that the swine plagues of Great Britain and the United States are one and the same disease.

In order to keep up a certain connection in this sketch, it is now necessary to refer to the work of Professor James Law, of Cornell University, Ithaca, N. Y. Prof. Law worked under the auspices of the Agricultural Department of the United States, and his results will be found in those reports. He followed Klein in his conclusions as to the etiology of swine plague, but, unlike Klein, does not furnish us with either a description or illustrations showing that he ever saw the true germ of swine plague. On the other hand, like Klein, he was somewhat successful in his inoculation experiments with cultivated and other material; hence, we must conclude that he also had the true germ in that material, but failed to recognize it. He says:

"This affection is characterized, perhaps (!) most important of all, by the presence of colonies of minute globular micrococci in the various seats of morbid change." p. 378, report 1878.

In support of the hypothetical etiological connection of these micrococci, which, from the language used I am led to think Dr. Law looked upon as the cause of swine plague, he quotes Dr. Klein, Eng., as follows:

"In 1877 Klein cultivated the micrococcus for seven successive generations * * * and finally inoculated the product of the fifth and seventh generations successfully in two pigs, which seems to establish that these microphytes are the ultimate cause of this disease." *Ibid.*, p. 378.

I regret to have to call attention to the direct contradiction of the above statement by Dr. Law himself, only three pages further on in the same report, where he says:

"From the cultivations of the fifth and seventh days respectively a drop was taken and two pigs were successfully inoculated.

"In the cultivations of each day were found myriads of bacilli, but no other organization [it should be organism—B.], and thus Klein was the first to show that the bacillus is the probable cause of this disease." *Ibid.*, p. 1881.

In the report of 1880-81 Law says:

"By a parity of reasoning it is probable that the swine plague, which is presumed to be caused like anthrax, by bacterian infection," etc.

It will thus be seen that Dr. Law has added nothing to the question, and hence we can leave him.

DR. H. J. DETMERS THE FIRST DISCOVERER OF THE GERM OF SWINE PLAGUE.

The first person to really discover the germ of swine plague was Dr. H. J. Detmers, at present professor of Veterinary Medicine in the State University, Columbus, Ohio. I have previously alluded to the success which has met Mr. Salmon's endeavors to "stamp out" Dr. Detmers and rob him of every credit for the really remarkable work done by him. Dr. Detmers' communications are to be found in the United States Agricultural Reports, 1878, 1879, and 1880-81. In the former report he mistook, through insufficient instruments, the micro-etiological organism of swine plague for a rod, to which he gave the name of bacillus suis, but in that of 1880-81, with better instruments and more experience, he gave a description, which, if not entirely correct, still is sufficiently positive to show that he did have the correct organism under observation and was endeavoring to describe it.

Detmers says:

"As to a proper generic name for the swine plague schizophytæ I am at a loss * * * They are not bacteria—by which he means rods—because the single cells are round." [Of this even he does not feel certain, for he immediately says:] "They can hardly be considered micrococci, because in their developed form they are bispherical." p. 185, Report 1880–81.

Detmers further says :

"The swine plague schizophytæ present themselves in different shape and form. The simplest form, it seems, is the micrococcus, a small round globule, which strongly refracts the light. The second form is bispherical. [This was his great error, as this is the first or mature form of the organism—B.] The globular cell, micrococcus, has duplicated itself. The globular or spherical cell, or micrococcus grows and becomes somewhat oval, in shape, but keeps on growing, while the indentations becomes deeper, still its length is about twice its width and its shape bispherical. For some time, however, the bilateral indentation does not effect a complete separation ; a connection between the two spherical cells remains sometimes only for a short time, sometimes longer. The bilateral indentation becomes deeper, while at the same time the single cells commence to grow and assume a somewhat oval shape, and in both, another bilateral indentation becomes visible. Meanwhile the separation in the middle becomes perfect, and soon one bispherical cell has developed into two bispherical cells or micrococci, which are yet slightly connected ; at any rate they remain together, although the separation appears to be perfect, as each cell presents its own outline." Ibid., p. 187.

My attention was called to a later publication upon the subject by Dr. Detmers, in the *Naturalist*, Vol. 16, by Dr. J. M. Heard, V.S., of New York City, and I am indebted to Prof. C. E. Bessey, dean of the Industrial College of the University, for the use of the volume in question.

Detmers considered it, in this paper, to be a micrococcus, and mistook this, the vegetative, for the mature form, and the mature form for the vegetative.

Here he says :

"The swine plague schizophytæ present themselves in three and probably four, and even five different forms. As to three different forms I am certain, but as to the fourth and possibly fifth I am not positive."

Then comes the first error in observation.

Detmers continues :

"The form to begin with is that of a very minute spherical body—a micrococcus. It is invariably present in the blood and blood serum in all morbid tissues and can be conveniently examined with high power objectives, when fresh. It probably is not necessary to state that the micrococci of swine plague, being spherical, do not present any characteristic difference from other micrococci, if the latter happen to be about the same size as the former."

Detmers continues:

"Still differences can be observed if the micrococci are kept under the microscope for some time (a few hours) at a suitable temperature. The swine plague micrococci soon form zooglea masses or aggregate in clusters and become imbedded in a comparatively viscid substance. While thus imbedded they soon commence to duplicate by growing in two opposite directions and at the same time become contracted in the middle. This contraction gradually becomes plainer and plainer and increases in the same degree in which the micrococcus is growing in length, till finally the latter presents the appearance of two closely connected spherical bodies without any visible partition and somewhat resembling the shape of a figure 8.

"At this stage the now bispherical micrococcus is about twice as long as its transverse diameter. (!!!)

In the interior of each spherical body a somewhat darker substance or a kind of nucleus can be observed. (!)

"This duplication or process of division which occurs in a large number of micrococci at the same time, it seems, finally breaks the glia, or viscous mass, which apparently holds the micrococcus clusters together; the micrococci, many, or perhaps most of them, are now bispherical [the real mature germ of the swine plague—B.] and some, yet single, become free and make their exit."—p. 200-201.

On plate III., figure 1, of this report, may be seen an illustration copied from the *American Naturalist*, p. 200, which represents Detmer's views of the appearance of this organism as it then appeared to him. It is easily to be seen that they are not "micrococci" as he claims; nor are they figure 8 organisms, with constriction in the middle as he pictures, yet that he meant to illustrate the real object there is no question, as I have personally seen his slides of the same, and he now illustrates those slides in Fig. 2, Plate III. Compare the latter with my own illustrations, Figs. 3 and 4, Plate III.

While there are many misconceptions in Dr. Detmers' descriptions of the biological appearances of the micro-organism of swine plague, still there is no doubt that he frequently had it under observation in

1880, though I very much doubt if he had it in a pure culture, unless by accident, from the descriptions which he, himself, gives of his methods of making his cultivations, such as :

“I charged an ounce of fresh milk, just drawn from the cow, with a mere speck of the proliferous growth of the stomach of Mr. L’s pig. The milk thus charged and contained in a perfectly clean two ounce vial, closed by a tightly fitting glass stopper, was kept at a constant temperature of from 99 to 100 degrees Fahr.”—p. 378, Report of the Department of Agriculture, 1879.

Anyone in the least acquainted with the methods of obtaining pure cultivations of micro-organisms will see that it would be next to impossible to obtain the same in any such way. Such a medium as “milk just drawn from the cow,” invariably contains a mixture of germ life. There is no mention made of the preparatory cleansing and disinfection of the teats of the cow or the hands of the milker even, and the same is true of the proper sterilization of the “perfectly clean two ounce vial.”

The apparent crudeness of Dr. Detmers’ methods of investigation must not be attributed to any want of knowledge on his part, for it was really magnificent, considering the state of our knowledge, on his part, at the time he worked, and especially when we consider the condition of instruction at the German veterinary schools at the time he studied. My admiration for Dr. Detmers’ scientific abilities is still more augmented when I consider the very poor laboratorial conveniences that he enjoyed.

In introducing some quotations of his work, which at first sight would appear to be with the purpose of detracting from its value, I desire to say that my real purpose is quite the contrary. These quotations have a twofold value :

1. They show how great the quality of his work was.
2. They show how much we have improved in the methods of bacteriological investigation, and are also instructive as showing how errors may be avoided in the future.

Oil immersion lenses and the Abbe condensor had not been perfected at that time, hence Detmers did his work with water immersion lenses, and he tells us with the simplicity of an honest man that one day :

“January 27, in the afternoon, I filtered some pulmonal exuda-

tion from a pig that had died of swine plague, through several pieces of paper, for the purpose of freeing it from the bacillus germs which it contained. The filtering was done on a small table in the corner of the room, and the apparatus was left standing on that table with the wet papers (4) in the funnel after the filtrate had been removed. In the evening the latter was examined under the microscope, on another table in the opposite part of the room, and as my two highest objectives are immersion lenses, I had to use water, and had a tumbler full of clean water on my table just drawn from the well. When through with my work, instead of pouring the water out, I placed the tumbler on another table, about four feet distant from the filtering apparatus.

"Next morning I went to Chicago to return on the 30th. In Chicago I procured a new immersion lense, and not intending to examine but a test object, I did not go for fresh water but used a drop of the water in the tumbler, which was standing exactly where I placed it.

"While adjusting the focus I discovered the water, which I knew had been absolutely free from organic matter, was swarming with bacilli and bacillus germs of the same kind as those in the pulmonal effusion."—p. 383, *Ibid.*, 1879.

Detmers then assumes that, as the above mentioned filter paper had dried, and during his three days' absence that the germ in or on it had got into the air of his work room and fallen into the tumbler of water, which had been drawn from the well and which to a dead certainty contained micro-organisms when it was drawn: it was then placed in a tumbler originally used for the purpose, which also had the same organisms on its sides to an equal certainty, and left exposed to the air of his ordinary work room for three days, which was still more certainly contaminated with any desired mixture of such organisms.

Again he says:

"On September 20, Prof. B. charged two drachms of fresh cow milk with a mere speck, smaller than a pin's head, of a decaying morbid growth, or ulcerous tumor of the cæcum of pig No. 5, and kept the vial closed at a temperature of 92 degrees F.

"On the evening of September 23, the milk was examined under the microscope, and found to contain numerous bacillus suis and bacillus germs, the same as found in the blood serum or exudations of diseased lungs, and in the decaying substance of the intestinal growths."

Another example will suffice:

"On June 10 I took two perfectly clean four ounce vials and put in each three ounces of clean well water, in which no bacteria or any living thing could be found. In one vial marked No. 1, I put half a drop of the fresh pulmonal exudation of a hog that had died of swine plague. p. 385, Report 1879."

The more one reflects upon such a methodic the more must be his astonishment that Dr. Detmers should arrive at such comparatively accurate ideas of the biological conditions presented by the bacterium of the American swine plague. As has been said, a pure cultivation of this organism, under such organisms, can have only been an accidental occurrence.

Even the material which I have quoted from Dr. Detmers' publications has been very carefully selected from among many vague assertions, and in some cases absolute contradictions, as may be seen from the following passage.

"The spherical, or single micrococci, undergo their first change and develop into bispherical bodies, till the glia breaks open, when a great many bispherical schizophytæ, and also some of the spherical bodies become free. The former (the bispherical bodies) thus freed, very soon commence to multiply by fission, but this process results in the production of bispherical, not spherical cells, or micrococci, the latter must have another origin." p. 188, Report 1880.

No further evidence is necessary to show that while Detmers at many times describes bio-phenomena connected with the germ of swine plague, though, at the same time, he had no distinct idea of the real vegetative pneumonia of the object he was describing.

In the above quotation he contradicts himself in the most startling manner.

First he tells us that :

"The spherical cells, or single micrococci, undergo their first change, and develop into bispherical bodies," which is absolutely correct, and then he says they do no such thing by saying that "these spherical cells or micrococci must have same other origin."

He missed the first stage of development, that from the mature germ or bispherical bodies, into micrococci, and describes the second, or the development of micrococci into the mature germ.

Detmers' uncertainty at this time so deceived Loeffler (Berlin) that he failed to recognize any resemblance between the micro-organism discovered by him in a swine disease of Germany, and that seen by Detmers in 1880.

PERSONAL OBSERVATIONS UPON THE GERM OF SWINE PLAGUE.

My first examinations of swine that had died of swine plague in Nebraska were made in July, 1886, and the reader can well judge of my surprise when I found, instead of a "micrococcus," a similar organism to that described and pictured by Dr. Detmers. To use a popular expression, "I could scarcely believe my own eyes." Since that time my necroscopical investigations have extended to over five hundred hogs that have either died or been killed on account of swine plague, and beyond that I have examined an immense amount of material sent me from adjoining states, from swine that were reported to have perished from the same disease, and in no single case have I failed to find this one micro-organism, which was first seen by Klein, in England, in 1876, though not recognized as a distinct species of germ life; then discovered and pretty well described by Detmers, in this country, in 1880; discovered by me in the tissues of an English hog that had died of swine plague in October, 1886, as well as re-discovered by me in this country prior to that date; discovered as the cause of swine plague in France, in July, 1887, by Cornil and Chantemesse, whose observations find later endorsement by Rietsch, Jobert, and Martinaud, all of which statements will be fully proven before this report is closed.

In no case have I been enabled to find a "micrococcus." With about one hundred autopsies at my command, all of which had given but one result, the reader can judge of my surprise upon receiving Mr. Salmon's report, issued 1886, in which he said, "we no longer consider a micrococcus to be cause of all outbreaks of swine plague," and then described an organism as the cause of that disease, which common sense and experience at once shew me could have no existence at all. As this part of the subject has been already fully discussed, we will at once proceed to that of the

TRUE AND ONLY GERM OF SWINE PLAGUE.

Before entering upon this side of the question it seems to me well to call attention to

THE MOST PRACTICAL METHOD OF OBTAINING PURE CULTIVATIONS FROM THE TISSUES OF DISEASED ANIMALS.

Pathological exudations, such as used by Dr. Detmers, serve very well to inoculate animals with, either to test the character of disease,

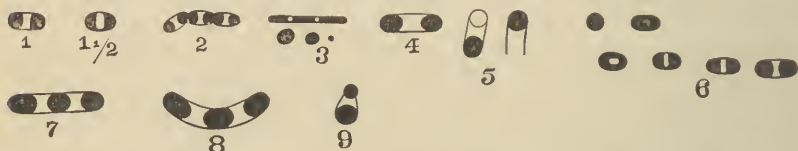
and, if contagious or infectious, to obtain, in most cases, absolutely pure cultivations of the germ by inoculating some sterilized gelatinous medium with the substance of some solid organ with every precaution against contamination by adventitious germs for the purpose of comparison. At the same time some small animal should also be inoculated from the same organs. When possible, the animal from which such material is taken should be killed rather than allowed to die. The cultures soon grow. They are to be examined and the pure ones used for the successive cultivation of any desired number of generations. At the same time the growth of the organism may also be tested in and upon any other media than the one first used. For primary cultivation agar-agar and sterilized blood serum are most advisable, as they permit the use of the thermostat. In the meantime the animals inoculated may become ill and killed, and tubes inoculated from their blood and tissues under due precautions; the resulting developments are then to be compared with those first obtained by microscopical examination and their deportment in the various media of cultivation. By this method one is enabled to get the organism in a clear and isolated form at a much less expense to the eyes and time than in any other.

Animals are then to be inoculated from the cultures, especially the species in which the disease occurs under natural conditions and if the same clinical and necroscopical phenomena result under these experimental conditions and the same germ can again be cultivated and is invariably found in the blood and tissue, its pathogenetic history has been in part discovered. Cultivations should be made from every animal inoculated, but in the future, when making field autopsies, it will suffice to consider the natural phenomena and to examine the blood and fluids by object glass specimens. In obtaining material for cultivations from autopsies made in the field, it is best to have two one gallon pails, made of heavy tin with tightly fitting covers. One of these should be kept full of a five per cent solution of carbolic acid and taken to the field in that condition. In the other should be a number of perfectly clean, large napkins soaking in the same solution, which should also be taken to the field in the same manner. The first pail should be emptied as soon as the operator is ready to remove the organs, which should be done as rapidly as possible; an assistant should be on hand and as soon as the organ is free, immediately wrap

it up in one of the above mentioned cloths which he has previously wrung out, and so on with each organ. This serves to sterilize the outside of the organs, and they are ready to obtain cultures from, on arrival at the laboratory, which must be done with all the precaution of hot knives, etc.

The plate culture isolation method can also be used at any time, but the above is far more practical for primary studies.

MORPHO-BIOLOGICAL CHARACTERISTICS OF THE GERMS OF SWINE PLAGUE.



Diagrammatic Illustration of the phases of development of the Bacterium of Swine Plague
See also Plate 3, Fig. 4.

The micro-etiological organism is neither to be looked upon as a bacillus, or micrococcus. In its mature form it is not round like the latter, or rod-shaped, like the former. In tissues, and occasionally in cultures, it sometimes develops in long threads composed of individual segments. It belongs to that intermediate group to which the name bacteria has been given.

The above classification is not in accord with the opinion of all observers, though agreeing with that of the majority.

In a series of articles in the *Berliner Klinische Wochenschrift*, 1886, Nos. 44, 45, 46, and 47, Hueppe (one of the most noted authors upon pathogenetic micro-organisms in Germany) has taken the etiology of the German swine plague into consideration, and promulgated views of generalization which I scarcely think are warranted by the existing facts.

The articles in question are upon the "Wild seuche," a peculiar infectious disease that attacks the deer tribe and cattle and swine, under natural conditions, but not sheep, and which has been transmitted to horses and the smaller animals generally used in experimentation.

The Germans speak of the deer as "Wild," and having no English word to express the meaning, I shall use the word "Wild seuche" in the following pages. Hueppe's hypothesis with reference to the Ger-

man swine plague, while probably correct as to that disease, will not be found to be equally applicable to the disease in this country.

Of the micro-etiological organism of the "Wild seuche," Hueppe says:

"The bacteria appear as short rods—stabeihen—in the blood, being two or three times as long as broad, and have distinctly rounded ends, markedly colored poles, and a clear, uncolored middle-piece; four of these objects correspond to the diameter of a red blood cell.

"Upon cultivating this organism in gelatine, they appear as isolated colonies, or they coalesce and form a grayish-white line, according to the quantity of material introduced on the wire. The edges of the canal are formed by the finest of isolated colonies. On the surface of the gelatine, which never becomes fluid, is formed a white circumscribed growth. Their development upon agar-agar is similar, the color being more of a grayish-white. Upon blood serum they form a fine transparent, opalescent coating. In bouillon, a cloudiness first occurs, followed by the precipitation of the objects to the bottom of the vessel, where they form a grayish-yellow mass. Upon potatoes they form a grayish-white coating."

Having given the above description of the growth of the organism of the "Wild seuche," Hueppe says:

"I look upon the vegetative form of this organism, in cultures and in the blood, as resembling cocci, according to their stage of development, [and also as to whether one sees them end on or not—B.] they present themselves to the eye, as round, or slightly elongated, ellipsoid bodies, which take up the coloring material in all parts of the body.

"This form soon extends itself to a shorter, or longer object, with distinctly rounded ends. The plasma of these short objects differentiates within the capsule, and isolates itself at either pole before fission takes place, while the capsule still retains the form of the short rod. It finally separates into two young roundish cells. According to the rapidity of growth and the age of the culture, the numerical relations of the different morphological appearances of this organism may vary, sometimes one form and then another predominating in the cultures. I have seen the short roundish homogeneous coloring rods in the blood of animals. They do not group themselves into chains or zooglea masses.

"The vegetative form must be looked upon as the coccus form of this organism, which does not suffer any material change of definition when we now and again find somewhat longer rod-like forms, and we must therefore credit this organism to the species micrococcus."

To call a mature micro-organism, as described by myself, a "micrococcus," because it passes through a coccoid-form in its develop-

ment, or has such a form in an embryonal condition, is as physiologically logical as it would be to call an ovum a man.

The two objects bear an equally exact relation to their respective mature forms.

In the earlier days of bacteriological research we described cocci as round, diplo, oval, or oblong or strepto-cocci, according as they presented themselves to the eye of the observer, but I defy any mortal human with honest eyes, and what is more rare, a logical and honest brain, to make a "micrococcus" out of "bacteria which appear as short rods (stäbchen) being two or three times as long as broad, and which markedly color at the poles with a clear middle piece."

It was a practical stroke of genius when the greatest of all pathogenetic bacteriologists, Robert Koch, relieved us of much difficulty by classifying the micro-organisms as :

1. Cocci—Absolutely round objects—not spores—that color homogeneously throughout.

2. Bacteria—Ovoid organisms, the longitudinal diameter of which exceeds the transverse.

3. Baccilli—rods.

4. The twisted organisms.

Now, here comes Hneppe, a most praiseworthy author and accredited observer, and throws the whole question into such chaotic confusion, that we can no longer make ourselves understood by the use of a single word, but must add a detailed description, in such case, in order that other observers may comprehend our meaning, especially when referring to past work.

In his opening remarks upon the morphology of the micro-organisms of the "Wild seuche," Hneppe distinctly says that, "Im Bluterscheint ein grosser Theil der Bacteria als Kurzes Stäbchen welches 2 bis 3 mal langer wie breit ist," a translation of which has been already given, but repeated reads, "in blood a greater part of the bacteria appear as short rods—stäbchen—which are two or three times as long as wide."

How any intelligent and educated observer can transform an object "two to three times longer than wide" into a "coccus," or call it a "short rod" in one place; and then say, that the same object should be called a "micrococcus," ("Wir müssen die Bakterien der Gattung Mikrooccus zuweisen") passes my comprehension.

Hueppe certainly understands the use of his own language, and I have no very insignificant knowledge of the same, yet in either English or German it is a *contradictio ad absurdum* to say that a "Stäbchen" (a short rod) is a "micrococcus" or round object.

No person can make a micrococcus out of this object, nor should any reflecting observer attempt to define or classify a matured object by any intermediate stage in its existence.

It would be equally logical and scientific to call the enclosed and comatose chrysalis a butterfly.

The micro-organism of American swine plague is very minute, and requires at least one-eighteenth oil immersion lense aided by the Abbe condensor in order to study its peculiarities successfully. My work has been with an instrument made expressly by E. Leitz, of Wetzlar, Germany, which, while not costing as much, was proved by the most exacting tests to be equal to the best Zeiss; it is provided with a one-twelfth, one-eighteenth, one-twentieth oil immersion, the latter of which I have used in these studies.

For a long time I have been of the opinion that we are not exact enough in our differential choice of language when speaking of the phenomena of micro-organismal life, especially with regard to the differentiation between morphological and biological phenomena.

Morphological phenomena have reference to shape, size, outline, alone.

Biological to everything else connected with these organisms.

The mistake is too often made of speaking of the appearances of the micro-organisms, when colored, as if they were morphological appearances only, whereas they are unquestionably of a biological, chemical character, also, dependent on the chemical affinities of certain parts of the protoplasm for the different coloring materials. While unquestionably, as in the case of the American and German swine plague, the Wild Seuche of Germany, and rabbit septicæmia, and those of some other diseases, this coloring reaction of the germ differentiates its protoplasm into two distinctly separate materials chemically, and this differentiation manifests itself to the eye of the observer in a certain morphological sense, still the phenomena are as much of a biological as a morphological nature.

Again, the artificial cultivations of micro-organism in or upon different media have their essential morpho-biological phenomena, in that

the cultivations frequently assume certain characteristic forms in some special medium.

The condition of biological research with regard to the pathogenetic micro-organisms is fast getting so contradictory that the investigators will soon have to come to some definite understanding as to the technical meaning of the words in order to understand one another. In no sense is this fact so apparent as in regard to the ovoid organisms which seem to belong to the class of diseases which will eventually be known as the infectious extra-organismal septicæmiæ.

The micro-organism of the true swine plague is then a bacterium in its mature form. It is not a micrococcus.

It is ovoid, being at least twice as long as wide when fully developed, its length as a mature individual being about one-sixth the diameter of the red blood cell of a hog when examined in freshly drawn blood under the microscope, and care is taken that no atmospheric or chemical influences interfere with the morphology of the blood cell.

It colors best in methylen-blue and methyl-violet, next best in the gentian-violet and methylen-green, also very well in the other violets, especially in a variety known as Hoff's violet, but not as well as many other organisms in fuchsine. As has been pointed out by Loeffler and others in Germany, the coloring capacity of many of these dyes is increased by adding to saturated solutions of the same in the coloring glass an equal amount of a solution of caustic potash 1:10,000 aqua.

The same must be filtered every time before using.

In coloring this organism the result will somewhat depend upon the length of time the covering glass specimen is exposed to the action of the coloring material, and the same is true with regard to the action of alcohol in the decolorizing of tissue specimens. Sometimes they do not color as well as at others, even when fresh covering-glass specimens are made and exactly the same tinctures used. Why, I do not know.

When not too intensely colored its protoplasm will be found to consist of two chemically different materials, which differentiate themselves over the body of the cell so that the two poles or ends are of a more or less intense blue, violet, green, or red color, according to the tincture used, while the center of the body remains uncolored. That

the outer cuticle or capsule of this organism is composed of the same chemical elements as the plasma of its poles, is to be seen from the fact that a delicate line of the same color extends from each of the colored poles along the sides of the object to the other, embracing the uncolored substance or middle of the body. Figures 1 and 1½, Plate 3, Figs. 2 and 3.

The above description depends upon the germ presenting itself to the eye in an exact horizontal position; that is lying straight on its horizontal axis. If, however, it be turned a little one way or the other, on its horizontal axis, numerous specimens will be seen where the white belt does not extend entirely across the object, as above described, but seems to be limited, more or less, to one side and more of the colored substance will be seen on the opposite side, than under general circumstances, or, perhaps better, by exact inspection. Fig. 1½. At first I mistook the appearance for the accumulation of the uncolored substance in this way during the process of its secretion from the colored ends which I take to be the method by which this non-coloring material is produced. I believe that one of the best ways to instruct others is to chronicle our errors and explain them.

Hence, I have done so in the preparation of this manuscript. More mature reflection has shown me that the above explanation is partially or wholly incorrect. The capsule of these micro-organisms has the same chemical composition as the pole ends, because it also colors somewhat under the same application of the tinction. Now, why does it not show the same intensity of coloring? The only answer is, that this capsule being very thin cannot take up as much color as the more dense pole ends; but being so thin by the same amount of exposure does not show any color when the middle of the object is looked directly down upon, but when the eye strikes the sides of the object, then we look through more material, and, hence see more color. Just as when we look at a piece of window glass, or a good glass slide. If we look directly through it, it is colorless, but if we turn it on edge and look at it, it has a more or less green shade, according to the quality of the glass. So according to the amount of exposure to the tinction, when not carried so far as to color the whole body of the germ, we have more or less visible coloring of the capsule, which can only be seen when we look through a considerable extent of substance, that is, on the sides of the object

Again, we may see two or three objects united together, all presenting the normal characteristics of full maturity. I have never yet seen more than three of these germs connected together (Fig. 2.) in fresh cultures, but in older ones and on white of eggs they develop quite long threads made up of segments. In general they either appear single or in pairs. In very old cultures these micro-organisms become thinner, more rod-like, and color more diffusely with the same degree of exposure to the tinction, and the white substance is either not visible at all or very faint. Fig. 3. Again, such old cultures are very replete in apparent micrococci of various dimensions which might lead one into the error of thinking that his cultures had become polluted. I call this last condition that of coccoid degeneration. Fig. 3. Or, we may see unusually long objects, the longitudinal diameter being twice or three times that of the mature organism, and the white, or uncolored, substance occupying a corresponding extensive amount of space, while the dark, or colored, ends may be somewhat larger or of the same size as those of the mature object. This condition represents the first step in the development of these organisms; that is, they become longer and more of this white substance is secreted. Fig. 4.

The next step in the process of vegetative development is the separation of one of the pole or coccoid ends, which then becomes free, and for a moment is exactly round like a coccus; and, as in a hanging drop culture (to which I always add a very small amount of an aqueous coloring solution), one will naturally see a very large number of these coccoid objects, on account of the fact that each individual present is continually going through the same process of multiplication. See Fig. 4, Plate III. Here, again, one may see a condition or phenomenon that might be misleading. One of the coccoid ends having been separated, the other may still remain connected with the white material, and as evidence that the colored ends have a greater degree of specific gravity as well as chemical composition, you will see in the continual tumbling about, and turning over of these objects, a white, round, or nearly so, colorless object directly under the eye, or numbers of these objects. When the germs in such a hanging drop culture have died from want of a sufficiency of nutrient material, you may see a large number of these objects, which could be easily mistaken for spores; but if we inoculate a new hanging drop culture from the same material used to

prepare the former, it will be found impossible to fall into any such serious error, for it will be easily seen that these non-colored, refracting points keep continually going out of sight, their place being taken by the coccoid, non-refracting point still attached to the other end of the white substance, and by watching one and the same organism in its continual turning over, first one appearance and then the other will be presented to the eye until the second coccoid end has become detached. Fig. 5.

What becomes of the uncolored transparent middle piece?

I do not know.

It appears, however, as if it underwent an almost immediate process of dissolution the moment it has become free from both of its polar attachments. That this substance does not represent a spore condition or have any relation to spores, is, to my mind, entirely beyond all question, as I have searched most diligently for spores in old and fresh cultures, and others made at all kinds of temperatures within the biological limits of these organisms, my search being inspired by the description of what I pronounce a forgery, of a germ (which represents a spore), as the cause of swine plague, by Mr. Salmon, in 1885, and again in 1886, as the cause of an assumed porcine pest, to which Mr. Salmon now gives the name of "hog cholera." This Salmon object does not exist, never has existed, and never will have any etiological connection with the American swine plague, as has been most conclusively demonstrated in this report.

In my first published description of the micro-organism of the swine plague I gave an erroneous description of the manner in which the coccoid ends became freed from the white or connective substance. This white, non-refracting, uncolorable material does not become extended to nothing and then break in two, leaving the coccoid ends with a delicate, colorless flagellum, or spermatozoid tail, temporarily attached to one side, as I then said, and as Detmers described it in 1880; but the separation of these ends is direct and by sharp segmentation. Were it otherwise we could not see the sporoid, colorless ends of so many of these germs, when freed from one of their pole ends.

These objects being so exceedingly minute, it takes some time to educate the eye so that one can perceive every phase of development. There are days when one cannot study them continuously at all. The best way to study hanging drop cultures, when one desires to spend

several hours over them, is to first make some cover-glass specimens of the same material, or take any other slides of an object of the same size and form, and observe such for a time, thus preparing the eye to see what you want to see in the living, developing organism. Unless this is done, some very essential points will be surely missed and some preventable error fallen into.

With anything less than a power of 800 diameters, no one should attempt to study these organisms, and then only when aided by the best of Abbe's condensers and oil immersion lenses.

We left our studies with the mature object proliferated into its first distinct stage of vegetative differentiation. We have two coccoid objects before us. That is, two round objects, their diameters being the same in any direction. If colored, they color throughout, that is, diffusely.

Were these objects to remain in this condition they would be indeed "micrococci." They do not, however. They almost immediately begin to increase in a longitudinal direction, but in this condition they still stain diffusely. In my first description of the swine plague germ, I said that the next biological phenomenon was the appearance of a delicate white line separating this ovoid object into two halves. The above, while not exactly an erroneous description, is certainly anticipated by another phenomenon in the evolutionary development of this coccoid, diffusely coloring object into the mature form of any of this class of germs. That this white, non-coloring substance is a secretion of the two poles or coccoid ends of these "belted" germs, is beyond all question, as well as that it has a different chemical composition.

These two facts, when taken together with the previously stated one, that the white substance almost, if not instantly, disappears from view the moment both of the coccoid pole ends have become shed off, segmented, leads directly to the following hypotheses: May not this white substance constitute, in part at least, the ptomain, or essential poisonous, pathogenetic principle in connection with these "belted" septicæmic germs, and may not this process of the immediate dissolution of this white substance be the means by which the ptomain gets into solution and then permeates the fluid cultivating media and the blood? Or is this substance fluid, and by the separation of the ends does it thus escape?

To my mind, these suppositions are worthy of consideration. The

fact that we can find no evidence of the development of permanent spores by these germs, and that this white substance is a secretion of the pole ends, goes largely to support these hypotheses.

The phenomenon above spoken of, as anticipating the formation of the segmenting white line which separates the two darker portions of these organisms is, that this white substance first appears in the center of the body of the dense, dark, ovoid object as the minutest of white specks, which gradually increases in size and quantity and extends across the entire object, the white line being at first broader in the middle, but gradually widening until it completely and clearly separates the two pole (coccoid) ends, and the mature object is again presented to our view. Fig. 6.

We have thus described the normal, or general, cycle of development of the micro-etiological organism of the swine plague, the "Wild seuche," and hen cholera, as well as rabbit septicæmia, Texas fever, and yellow fever, all of which diseases are caused by a member of this class of "belted" germs, and should be classed as extra-organismal, local, or land, septicæmiæ.

There are, however, other phases in the development of these germs of a bio-morphological character. For instance, as already said, we may see two or three individuals of the mature type together, Fig. 2, or we may find two apparently mature organisms inclosed in a common capsule, the two medial dark points, or poles, being in such close apposition that no line of demarcation or indentation of the capsule can be seen at this point, the whole outer surface being smooth. Fig. 7. On the other hand, the two lateral ends, or free poles, are separated by the normal quantity of white, non-colorable substance. Again, these diplo-bacteria may assume a curved or sausage shape, which we may sometimes see intimated in the single organism (mature). Fig. 8. At other times, though not very frequently, the germ may appear in nearly its normal form, but one pole (coccoid) end will be semi-segmented from its appositional end of the white substance by a constriction of the same at its line of attachment with the pole end. Fig. 9. This end will then be smaller than the opposite pole, thus giving a sort of pear shape to the entire organism; the small pole end is soon dropped, however, and becomes momentarily a free coccoid, and goes through the cycle of morpho-development already described; the same occurs with the other pole end.

This concludes my observations of the micro-morpho-biological phases presented by these micro-etiological organisms in the course of their development. There may be some minor phenomena that have escaped my attention, but I am very sure I have described all the essential points.

THE DEVELOPMENT OF THE SWINE PLAGUE GERM ON POTATOES.

When one reads the account of the investigations of Loeffler and Schütz, he will see that the former asserts that the germ discovered by him did not develop upon potatoes, not will not, but did not. Loeffler says:

“Anf gekochten Kartoffeln kamen sie bei vielfach wieder holten Uebertragungsversuchen nicht-zur Entwicklung.” (Arbeiten a.d. Kaiserlichen Gesundheitsamte 1886, p. 49.)

The germ of the German disease *will develop upon potatoes*, which assertion has been confirmed by Hueppe and by a personal letter from my friend, Professor Kitt, of Munich, dated September 17, 1887, in which he says: “Sie wachsen auf Kartoffel,” that is, grow on potatoes. Cornil and Chantemesse also make the same assertion for the organism discovered by them: “Sur la pomme der terre, culture abondante de couleur grise,” that is, they grow with a gray color. In no case have I failed in receiving a development upon potatoes with the germs of swine plague as I have found them in this country, and in all cases the development has assumed a grayish-brown, or peculiar coffee color, when the germs are reasonably fresh from diseased hogs, and the potatoes sufficiently cooked. This last is a very important factor.

THE DEPARTMENT OF THE GERMS OF SWINE PLAGUE IN BEEF INFUSION GELATINE.

In freshly made meat infusion peptonized gelatine the first seen bio-phenomena are somewhat dependent upon the size of the wire and the number of germs upon it when the medium is inoculated. If the wire is delicate, and the number of germs small that are attached to it, the individual colonies will develop as in the older gelatine, especially at first; but they soon grow larger and finally coalesce, forming a continuous development along the line of puncture. Along the sides of the same the small, individual colonies are indicated by

its delicate serrated appearance. I have seen this organism, when inoculated into old gelatine, retain the isolated colonial appearance for a period of two months without any attempt at coalescence between the colonies.

They never cause the gelatine to become fluid.

They are as anaerobic as aerobic; that is develop equally well with or without contact with the air. They develop as a sharply circumscribed coating of a grayish-white color upon the surface of freshly made gelatine, but not too profusely.

IS THE BACTERIUM OF SWINE PLAGUE MOTILE OR NOT?

This seems to be a question which has troubled the mind of Mr. Salmon very much indeed, and from reading his descriptions of this point we have seen that it moves at one time and not at another. The real reason of Mr. Salmon's myopia is to be sought in the fact that I insist that this micro-organism has motion, and he therefore must have it motionless, but at the same time knowing it has motion, in some way or other he frequently contradicts himself. He is simply trying to swallow that monstrous whale, his own inconsistencies. Another reason is, that Salmon falls back on Schütz to get support in favor of his assertion that swine plague (not Salmon's "hog cholera") is a pneumonia only, because Schütz has said that the "Schweine seuche" is an "infectious pneumonia." Salmon said that half of the American disease is the same, but to be original, adds "chronic" to it, so as to differ a little from Schütz. Naturally he borrows Schütz's germ for this disease of his, but does not know that Schütz himself was studying two different diseases, but did not recognize the fact. Having borrowed the Loeffler-Schütz germ (the whale becomes troublesome) it must deport itself just as they say, so it does not grow on potatoes, when seen through Americo-German glasses, but when grown on Yankee potatoes it does, for Salmon tells us how it differs from the other thing (hog cholera) when developed upon these media. Then comes the motion question. Salmon fails to see that Schütz does not say that it has no motion; he does not deny it motion, he simply says, "it has no independent motion." *Die Bakterien Keine selbständige Bewegung ausführen.* p. 381. Prof. Kitt, of Munich, writes me in identically the same words. As will be seen later on, Rietsch, of Marseilles, says these objects do move, and in a letter to Salmon says how

they differ in quantity of motion from some kind of a thing of which he sent Rietsch a culture. Swedish observers also see motion, though they deny any identity between their organism and that of Loeffler-Schütz. Now it is easily to be seen that here is no denial of motion, in fact motion is inferred by both Schütz and Kitt. Hence, the question narrows itself down to *what kind of motion?*

There is a phenomenon known to bacteriologists as "molecular or Brownian movement," which is a sort of oscillating motion, a dancing up and down, without change of place in fluids under the microscope of granular objects, when the object is so fixed that the surrounding air cannot act upon them; when it does there is a slight suction of currents present, and then there is also change of place.

Now, it is this motion which it seems to be the German observers attribute to these micro-organisms.

I cannot possibly agree with their conclusion. In a well-prepared hanging drop, and I have gone so far as to remove the grease extending beyond the edges of the covering glass and replace it with cement, the action of the surrounding air upon the drop is absolutely excluded, yet these objects move, and not in the Brownian sense, either. Their movements, however, are not the active ones of the micro-organisms provided with cilia-like ends; yet they change their place slowly; they disappear entirely out of sight, and frequently appear again at a short distance from the point where they disappeared; when two members are united together, they twist in various directions in their endeavors to separate, in the same manner that we twist a very tough piece of stick in order to separate the ends. Hence, I assert the bacteria of swine plague "do move," every and any authority to the contrary. I do not say this entirely of myself, for once and a while a competent botanical microscopist comes to see me, or even some good histologist, and in every case I have been interested to test them upon this point, and ask them as to whether they thought it was an independent or Brownian movement they were observing. The invariable answer has been, "He do move," and not one has considered it the "Brownian movement." And so, as the negro preacher said of the sun, we can close this question again with the words, "He do move."

PART II.

PATHOLOGICAL ANATOMY IN SWINE PLAGUE.

PATHOLOGICAL ANATOMY IN SWINE PLAGUE.

NECROSCOPICAL OBSERVATIONS UPON HOGS DISEASED WITH SWINE PLAGUE.

The first diseased hogs that I saw in Nebraska, which were in fact the first swine I ever saw having this disease, belonged to a Mr. Wagner, who lived about four miles north-east of the city of Lincoln. He had quite a large number sick at the time, and had already lost some. This was on June 14, 1886, and before I had any conveniences for the microscopic study of the disease, or for investigating experiments in a laboratory, so that nothing was done at this time towards discovering the germ, or micro-organism, which causes this terrible porcine malady. Mr. W. kindly gave me two sick hogs that were about one-third grown, which were taken to the experimental farm of the University for further observation. Both of these hogs were black in color, being from Poland-China stock crossed on less well-bred swine; they were weak and somewhat emaciated; both had a dry, distressing cough, and a watery discharge from the anus, which was of a peculiar dirty yellowish-green color, and gave off an offensive odor, which many say is so characteristic of this disease that they can diagnose it at some distance from the place where such swine are.

At the "College Farm" were a number of hogs which had had the swine plague the early part of the previous winter and had fully recovered from the attack. They were all somewhat stunted in their growth, from the effects of the disease. These hogs have been subjected to all sorts of experiments, which have been repeated again and again, in order to test the question whether hogs that have once been through the swine plague in a natural manner, and fully recovered, were, or would be, susceptible to a second infection, whether by exposure among large herds of diseased swine, or by means of artificial inoculation with a virus containing pure cultures of the germ of this disease. It may be well to barely mention, at this time, that these hogs have withstood every attempt to induce a second infection, though exposed to the most severe natural and artificial tests, and that they

are still alive and as well as any swine could possibly be, and that the sows among them have produced very fine litters of pigs. Hence, though having swine plague does generally interfere with the full development of pigs, it does not materially interfere with the reproductive qualities of the females.

The two Wagner hogs, being, as mentioned, decidedly sick, were placed (June 14, 1886) in newly built pens, in which were also placed one each of the white farm hogs that had previously had the disease.

On June 22, one of the Wagner hogs died. During its confinement at the farm it had been gradually growing worse, the choleroïd discharge becoming thinner and thinner and the evacuations more frequent; the cough also became more distressing, while the animal became excessively emaciated.

AUTOPSY. I.

Black hog, five months old; *no discoloration of the skin*; general atrophy of the sub-cutaneous fat-tissue; on cutting through the skin the blood that oozed out from the blood-vessels was of a dark, bluish-red color; it coagulated easily, but not so quickly as normal blood, and soon grew bright red in color upon exposure to the atmosphere.

The superficial inguinal glands were much swollen, and, externally, presented a peculiar mottled appearance, the darker portions being of a dense, dark, bluish-red color; the cut surface of these glands swelled up above the embracing capsule, and presented a moist and glistening appearance—œdematous; the general normal appearance of this surface was interrupted by numerous hemorrhagic centers in the cortex, sub-capsular, and in the interstitial spaces, which, being of the above mentioned dark, blue-red color, gave, when contrasted with the grayish color of the parenchymatous portions of the gland, that peculiar mottled appearance to which Prof. Walley (the accomplished principal of the justly celebrated Dick Veterinary College of Edinburg, Scotland) has given the name of “strawberry-like,” comparing it to the cut surface of a large strawberry. It may be remarked that this lesion of the lymph-glands will never be missed in any acute case of swine plague, but as it also occurs in other forms of septicæmia in swine, “Wild Sueche” cannot have the pathognomonic value I once attributed to it. In cases which have assumed a somewhat extended character, this condition of the lymph-glands may not be present, or more or less indistinct.

Thoracic Cavity:

No effusion present. Lungs somewhat hyperæmic; anterior lobes and lower portion of middle lobes solidified. Pleuræ normal. No effusion in the pericardial sack. Myocardium, or muscle-tissue of the heart, swollen, anæmic, opaque, yellowish-grey in color and very friable. On cutting across the diseased portions of the lungs, the cut surface was moist, and glistening in the freshly complicated portions, and of a general dark red color. The interlobular vessels, in these portions of the lungs, were densely engorged with blood, this condition extending into the surrounding tissues, which were more or less hyperæmic, but gradually lost itself as one approached the normal parts. In other portions of the diseased parts the entire lobuli were frequently anæmic, more or less dry, opaque, and of a yellowish-grey or grey-red color—caseous pneumonia; in these the walls of the bronchiales could only be distinguished by their whiter and more lustrous appearance; other lobuli were the seat of destructive processes which extended to the connective tissue septa, which separated them from adjoining lobuli; in this way small, irregularly outlined cavities were formed, which were filled with a more or less puriform material, which, in general, had a tendency to be of an aqueous character. The mucosa of the trachea and large air tubes was swollen; the larger vessels being strongly injected, with several hemorrhagic centers distributed through the substance of the membrane; the lower part of the trachea and larger tubes were filled with a fine, frothy material, having a somewhat yellowish color. The bronchial lymph-glands were intensely swollen and of a diffuse, dark red color externally, while internally they were moist and glistening, and presented the previously described mottled appearance, though the interstitial hemorrhages were far more excessive than those described in the external inguinal glands.

Abdominal Cavity:

Liver: swollen, edges rounded, external surface of peculiar mottled reddish color. Cut surface, anæmic, somewhat dry, of a yellowish-grey-red color. Acini distended; central and interacinous vessels compressed. Spleen: swollen in all directions; outside surface irregular and mottled by the interstitial tissue which shew through the capsule. Cut surface moist, glistening and full of a dark red fluid which oozed over it. Kidneys: somewhat swollen, capsule non-

adherent, external surface dotted by numerous dark red spots of the size of a pin's head. At first sight these spots appeared somewhat regular in size and more or less circumscribed, leading one to suppose that they represented the distended malpighian tufts, an error which several observers have fallen into. Closer inspection, however, demonstrates the fact that these dark red spots were not only very irregular in form, but that they varied much in size, and that, instead of being sharply circumscribed, they were diffuse in their outlines, and hence must be extravasations. The cortical substance of these organs was anæmic, opaque, yellowish-red-grey in color, and much swollen; not an indication of a blood-vessel could be seen, with the exception, that the previously mentioned dark red spots also dotted the cut surface of this portion of the kidneys. The medullary substance was of a dark red color, the vasa-recti signifying their presence by their still more pronounced color and injected condition. Lining of the pelvis of the kidney intensely swollen and the seat of more or less marked hemorrhages; it was coated with an undue amount of viscid material. Stomach: peritoneal covering somewhat swollen and clouded; vessels injected; partially filled with ingesta. Mucosa swollen and covered with a thick, viscid coating; towards the pylorus the mucosa was deeply stained with gall.

Intestines: The outside of the small intestine was of a diffuse, greyish-red color, clouded and somewhat swollen with an occasional vessel injected. That of the large intestine was of a greyish color, clouded and swollen. Duodenum and Jejunum: contents semi-fluid, and of a dirty yellowish color. Mucosa swollen, stained yellow by the gall, but interrupted by a few petechial hemorrhages. Ileum in the same condition, except that the general color of the mucosa was of a leaden-gray shade, which became more marked as the cæcum was approached; anterior to the ilco-cæcal valve was a small irregularly outlined ulceration with swollen edges. Large intestines: Contents fluid, and of a yellowish-green color and very offensive odor. Mucosa swollen throughout and of a leaden color, the diffuseness of which was interrupted by innumerable small black spots looking as if coal dust had been sprinkled over it before the somewhat viscid coating had been formed. No ulcerations present.

AUTOPSY NO. II.

Subject, a large hog about nine months old; color, black throughout; condition, fair. The tissues around the nose, lips, and sides of the head, as well as in the intermaxillary space, were much swollen; in some places the skin was eroded, and from these exuded a reddish, watery fluid; on cutting into these parts, the sub-cutis was found to be swollen and of a bluish-red color; they were filled with the same kind of fluid. The skin of the abdomen and inside of the fore-arms and thighs was of a lighter shade of black than the rest of the body; its regular color was interrupted by numerous diffuse, dark, bluish-red patches of varying dimensions. On section of the skin a dark, blue-red fluid issued from the cut blood-vessels which soon coagulated and became bright red upon contact with the air. The subcutaneous fat-tissue was the seat of numerous diffuse, dark, reddish patches, which corresponded in location to those previously mentioned in the skin of the abdomen.

Abdominal Cavity:

The peritoneal cavity contained about two quarts of a straw-colored fluid in which much flocculent material floated. The peritoncum, itself, was swollen and clouded; its generally grayish color being interrupted by immense numbers of bluish-black petechial spots of irregular contour. The large intestines were agglutinated together, and the meso-colon covered with a mass of viscid flocculent material. The surface of this intestine was very irregular, the haustra, or sacculations, being distinctly marked as smooth extensions separated by constricted, or indrawn, portions of the walls; these sacculated portions of the large intestine were each filled with some dense, hard material, which felt like medium-sized apples, being roundish in form. The small intestine presented a diffuse, reddish appearance, frequently interrupted by numerous dark red spots of irregular size. The mesenteric and inguinal lymph-glands were very much swollen, and presented a somewhat variegated external appearance, the usual greyish color being interrupted by numerous dark, blue-red spots of irregular form and size. The cut surface was moist and glistening, and swelled above the limits of the sectioned capsule; the general appearance was varied by many hemorrhagic centers, some being within the capsule and extending into the interstitial substance, giving them rather a V shape, while others were roundish or elongated, and situated in the same tissue and distributed through the substance of the gland.

Stomach: Peritoneal covering swollen, opaque; vessels injected with an occasional hemorrhagic center in its substance. Partially filled with a dirty, greenish mass and some half-digested corn. On opening this organ its cardiac portion was found to be covered with numerous papillary vegetations, which had a yellow color. The mucosa was excessively swollen, the rugæ (or folds) being very prominent; on the crest of these folds were numerous irregularly shaped ulcerations; hemorrhages of various dimensions were distributed throughout the mucosa, which was covered by a heavy coating of a viscid material which assumed a yellowish color in the vicinity of the pylorus.

Small Intestines: Contents of the duodenum fluid and of a greenish yellow color; mucosa very much swollen, and stained yellowish; numerous ecchymotic spots and diffuse hemorrhages in the substance of the membrane. Jejunum: contents semi-fluid and more greenish in color than in the duodenum; mucosa intensely swollen, glands very prominent; extensive and diffuse hemorrhages were to be seen in many places, some of these were so extensive as to remind one of the so-called "eel-skin" seen in the rinderpest of cattle in Europe, being of a dark, blue-black color. Ileum: presented a similar appearance; solitary follicles and Peyer's patches very prominent; many small hemorrhagic centers in the substance of the latter.

Large Intestines: Contents of the anterior portion semi-fluid and of a greenish-black color; it soon became more concentrated, and formed into large, hard balls of the same color, which completely filled the haustra and were closely attached to the mucosa by some viscid material. The entire mucosa of the large intestine was intensely swollen and of a diffuse, dull, leaden color, interrupted by numerous hemorrhages of varying form and dimensions, which were of a blackish-red color. The ileo-caecal valve was excessively swollen and extended for more than an inch in the lumen cavity of the cæcum; the mucosa covering it was much swollen, indurated, and of a dark coal-dust-like appearance, except on the apex of the valve, where it assumed more of a dirty greyish color, growing lighter from the circumference towards the center, or opening of the ileum into the cæcum; around the valve the mucosa was also excessively swollen, and the seat of extensive intra-mucosal hemorrhages, which gave to it a blackish-blue-red appearance; the membrane had not lost its luster in these parts, however. About six inches from the valve was a large,

ragged-edged ulceration of the same color; it was about one and a half inches in diameter, and had a crater-like formation, the base being also very ragged; outside of its intensely swollen and ragged edges was a sort of furrow, which separated it from the adjoining mucosa, which was much infiltrated and rose, umbus-like, around the ulceration, and was of the same dark, blue-red color. See Plate IX. The whole presented a decidedly wicked look. This was the only ulceration worthy of mention in the entire course of the large intestine, and is also of great value as an example of the effects of acute arterial embolism in the walls of the intestines; all indications of the so-called button-like neoplastic productions were wanting. The other portions of the mucosa were also much swollen and the seat of numerous small circumscribed and larger diffuse hemorrhages; numerous follicular ulcerations were present, some of them covered with a yellowish-grey, caseous coating, which left a congested surface when removed; the sub-mucous vessels were intensely gorged, and in the larger ones the blood had coagulated.

It must be here remarked, that more severely acute and excessive complications of the large intestine can scarcely be met with in a case of swine plague, and yet in this case, there was not an indication of choleroïd phenomena, either clinically or necroscopically, thus showing, that the intestinal lesions, in this disease, have no connection whatever with the choleroïd phenomena which have given it its popular name of "hog cholera." The real cause of these phenomena, when present, will be fully considered in another portion of this report; that is, in speaking of its pheuomenology from a general pathological point of view.

Kidneys: Both of these organs were more intensely swollen than we have ever met with in this disease, either before or since; seen through their capsules, they presented a peculiar putty-like color, which, taken in connection with their swollen condition, reminded one most strongly of the "large white kidney" of Bright, which indeed they were. These organs were so nearly alike in size that the measurements of one will answer for the other. They were 9 inches long, 3 wide, and one and a half inches thick in the middle of the organ. On cutting through their vascular connections, a large quantity of dark, blue-red blood oozed from the renal openings, especially of the ureters. Capsule non-adherent. Cortex intensely swollen, and anæmic; opaque and of

a dull, yellowish-grey color. Not an indication of a blood-vessel could be seen. Medullary substance was of a diffuse, dark, maroon-red color; the vasa-recti were only visible as the limits of the cortical substance were approached. The pelvic cavity was full of a mass of coagulated blood, which filled all its crevices and was intimately attached to its lining membrane, which was much swollen and the seat of numerous diffuse hemorrhages in its substance.

Liver: Excessively swollen; edges rounded; peritoneal covering normal, with the exception of a slight cloudiness, but through it could be seen numerous small, bluish-black spots of a more or less circumscribed character; but especially marked, however, were the greater number of yellowish-red, irregularly formed spots which were distributed all over the surface of the organ. Upon cross-section, the internal view which the liver presented corresponded somewhat with the external, there being almost innumerable small hemorrhagic centers in its substance, most of which were in the inter-acinous tissue, and many of which complicated the acini, completely destroying their characteristic appearance. Aside from this, however, the general appearance of the liver was anæmic, opaque, and of a greyish-yellow-red color; the acini were excessively distended in these portions, their central vessel being compressed and invisible. In many places whole groups of acini were so anæmic and had undergone such an excessive degree of fatty degeneration that they appeared almost semi-fluid and very fatty; these corresponded to the yellowish-red spots seen through the capsule. In others, that were not so severely complicated, their outlines were very distinctly marked by the distended gall ducts. In general, the interstitial tissue was swollen and œdematous. Gall-bladder distended with a viscid yellow material, having a tendency to a reddish-yellow color.

Spleen: Much swollen; 16 inches long; $3\frac{1}{2}$ wide at the superior end and only 1 inch at the inferior. The pulp of the organ was very full of blood and much degenerated, forming a dark, bluish-red semi-fluid mass; trabeculae swollen and distinctly visible through the capsule, giving to the organ a peculiar marbled appearance. Malpighian corpuscles swollen and distinctly visible.

Thoracic Cavity:

The right side of this cavity contained a large amount of a dark red fluid; the lungs were loosely attached to the ribs in many places,

while their base closely adhered to the diaphragm throughout its entire extent. The costal pleuræ were intensely swollen, rough where the lungs had been detached; numerous dark red spots of variable dimensions were to be seen in the membrane, some of them were circumscribed, while the larger ones disappeared diffusely in the surrounding tissue.

The surface of the lungs was roughened and thickened at those points where it had been attached to the costal pleuræ and marked by numerous dark red hemorrhagic centers of various shapes and extensions; many of these were more than superficial or sub-pleural, and occupied either the extent of an entire lobulus, or complicated several. The right lung was the seat of numerous large centers of consolidation, its base being one solid mass. Smaller ones were seated in the left lung. On cross-section of these parts an aqueous, bloody fluid oozed from the cut surface, but only from those parts which were extensively consolidated, and the adjoining excessively hyperæmic tissue; this peculiarity, that is, the limitation of the œlema pulmonum to the diseased portions of the lungs, has been met with in all such cases that I have seen in swine plague; collateral hyperæmia and œdema, so common in pneumonia in man, does not seem to occur in this disease. The only explanation, to my mind, of this peculiar phenomenon must be sought in the fact that, in such cases, obstruction of the circulation in the larger vessels leading to the left heart, by the bacteria and coagulation of the blood, and consequently the obstruction of the circulation occurs before the inflammatory process in the alveoli takes place, and hence, as the right heart is still driving the blood into these parts, that the terminal capillaries of this portion of the lungs become over-distended, and the changes in their walls, due to stagnation of the circulation, occurring, the aqueous portions of the blood must become extra-vas and accumulate in the alveoli (and interstitial tissues) with the products of the inflammation in the former. The cut surface of the solidified portions of the lungs presented a peculiar lustrous appearance, on account of its hyperæmie, œdematous condition; the interstitial spaces were especially marked by the distended condition of the blood-vessels, which were filled with coagulated blood, as well as their œdematous swollen condition; an examination of the coagula revealed, in this case, as well as every other of a similar character, the presence of innumerable bacteria which we have found specific to this

disease. To the casual observer this condition of the interstitial tissue of the lungs in swine plague looks very much like interstitial pneumonia. In fact, such mistakes have been made. A closer observation, however, will show that no neoplastic or indurative conditions are present in the interstitial tissue. All portions of the cut surface of the consolidated parts of the lungs did not present this lustrous appearance; it was interrupted by many localities having a dry surface and an opaque, grayish-yellow color, which were either limited to a single lobulus or complicated entire groups of lobuli; the material of which these centers was composed was dry and caseous; other lobuli were of a dense, dark red color, representing hemorrhagic infarction; still others were filled with an aqueous purulent material, their entire structure having disappeared, while in some cases there was destruction of the interstitial septa and several lobuli had thus been formed into one cavity.

The mucosa of the larynx, trachea, and bronchial tubes was very much swollen and dotted or variegated by numerous hemorrhagic centers of a petechial or diffuse character and varied dimensions; the vessels were also very much injected. The larger tubes were filled with a reddish-yellow frothy mass, while the smaller ones contained an aqueous material of a similar color.

The Heart: The pericardium was loosely attached to the appositional portions of the pulmonary pleura, and dotted with numerous ecchymotic spots. On opening this membrane its sack was found to contain about a tea-cupful of a reddish, watery fluid; its visceral portion was also dotted in the same manner. The myocardium was of an opaque anæmic, yellowish-red-gray color, and very friable; the right ventricle was distended and contained a semi-fluid, dark, blue-red material. The walls of the left ventricle were decidedly hypertrophied. The endocardium of both hearts presented numerous ecchymotic spots in both its auricular and ventricular extensions. The auricular-ventricular valves were shrunken and covered with numerous nodular neoplasms.

The bronchial lymphatic glands were enlarged and juicy, and presented the variegated external and internal appearance previously mentioned in the consideration of the other glands of the body due to sub-capsular and interstitial hemorrhages.

AUTOPSY No. III.

Three-quarters grown, black hog. Died sometime during the previous night. Rigor mortis moderate. Quite a number of diffusely terminating, dark, bluish-red spots to be seen in the skin of the abdomen, especially on the insides of the extremities and sub-pelvic region; these centers did not extend into the sub-cutis. On opening the abdomen the blood which flowed from the cut vessels was of a bluish-red color, soon coagulating and becoming bright red upon exposure to the atmosphere. The superficial inguinal glands were swollen, the cut surface of these organs being moist and glistening, but there were but a few circumscribed hemorrhages in the sub-capsular tissues and interstitial striæ.

Abdominal cavity:

No effusion present. The parietal peritoneum possessed its normal color and consistency, with the exception of a very slightly clouded and swollen condition. The vessels of the peritoneal covering of the large intestine were much injected, as well as those of the mesocolon and mesenterium. The convolutions of the large intestine were agglutinated together by a mass of viscid material in which many flocculi were observed. The small intestine was of a diffuse, pink-red color. The mesenterial lymph-glands were much swollen and the seat of numerous sub-capsular and interstitial hemorrhages.

The Liver: This organ was very much swollen, its edges being distinctly rounded. Even through the capsule the acini could be distinctly seen from their markedly distended character and yellowish-red appearance. Upon making a cross-section of this organ the parenchyma appeared anæmic and fatty; the general appearance being of a grey-yellowish-red color; the interstitial tissue was somewhat swollen, the gall ducts being frequently distended, and the interacinous vessels injected, while the central vessels were compressed and could only be seen with some difficulty. Gall-bladder distended and full of a yellowish-green, viscid material.

Spleen: This organ was swollen in all its dimensions; the superficial veins were somewhat engorged, and the trabeculæ distinctly visible through the capsule, which gave a somewhat variegated appearance to the external surface of the organ. On cutting it across, the parenchyma was found to be rather dry and bloodless, though some

blood oozed from the cut ends of the larger vessels; the malpighian bodies appeared to be somewhat swollen and were distinctly visible.

Kidneys: These organs were swollen. Capsule non-adherent. Cortical substance anæmic and of an opaque, yellowish-gray color, but occasionally interrupted by small, wedge-shaped centers in a condition of cloudy swelling only. The outer surface of the kidney was frequently marked by the presence of small, irregularly shaped, dark red centers of the size of a pin's head, but terminating diffusely. The medullary substance was somewhat swollen and reddened; the vasa-recti were distinctly marked and injected, their outlines being rendered still more distinct by the swollen condition and clouded gray color of the straight tubes. The pelvis displayed nothing abnormal with the exception of the slightly swollen and clouded condition of its lining membrane.

Stomach: Peritoneal covering clouded and swollen; many of the larger vessels injected. It was but partially filled with a dirty, greenish mass. Mucosa intensely swollen and gathered in folds along the base of the greater curvature; it was covered with a thick, viscid coating, beneath which were many circumscribed and diffuse hemorrhages, both superficial and deep; towards the pyloric termination the mucosa was stained of a bright, yellow color.

Duodenum: Contents fluid and of a yellowish-grey-green color. Mucosa swollen and stained yellow with occasional hemorrhagic centers to change the general monotony.

Jejunum and Ileum: Contents semi-fluid and of a dirty, green color. Mucosa swollen as above, with the exception that the hemorrhagic conditions were more extended and profuse, especially as the large intestine was approached.

Large Intestine: Contents pultaceous in the cæcum, but as the colon was reached it became more consistent and finally formed into hard round balls of a darkish green color; their consistency was even more dense in the rectum. The general appearance of the mucosa was leaden grey, with an occasional point where the vessels were injected, and where this condition of the capillaries gave a delicate, reddish blush to the membrane of a diffuse character; the membrane was but slightly swollen, except in the vicinity of the ileo-cæcal valve, which was considerably swollen; these ulcerative erosions about the size of the surface of a segmented pea were to be seen upon the crest of the valve, which

somewhat resembled a small hole punched into the tissue of the parts, but the edges were irregular; and the surrounding tissue was somewhat swollen so as to give a walled appearance to each of these erosions; the centre of these erosions was composed of a yellowish-grey, friable material which could be easily removed, and presented a ragged, ulcerated surface to view. Numerous smaller, follicular ulcerations were distributed through the cæcum and colon; in the latter they were less numerous but more extensive than in the former, but in both they were free from anything resembling a neoplastic or indurative character.

The Bladder: This organ was distended with urine which was clear, but on testing was found to contain considerable quantities of albumen.

Thoracic Cavity:

Both sides contained a considerable quantity of a reddish-yellow fluid. The lungs were adherent to the costal pleuræ of both sides, as well as to the pericardium and mediastinum, both bases were adherent to diaphragm in their full extent. In the thoracic effusion were floating large masses of a straw-colored gelatinous material. As said above, the pericardium was closely attached to the juxtapositional tissues; upon separating the adhesions numerous ecchymotic spots in both membranes came to view, the latter being much thickened and roughened and the superficial blood-vessels engorged. The pericardium itself was thickened to more than twice its natural dimension, its sack being filled with a yellow gelatinous material, through which ran many shreds of a fibrinous character from the external to the visceral, or cardiac, fold of the membrane; the visceral fold was also much thickened and the seat of numerous small hemorrhagic centers. The *myocardium* was anaemic and of a dull, yellowish-grey-red color, and opaque, and very friable. The entire *endocardium* was swollen, clouded, and the seat of numerous petechial and ecchymotic hemorrhages. Valves normal. Contents semi-fluid, with a small anti-mortem clot extending into the large vessels, especially in the right heart; in the right ventricle this clot was dark red at its base, and then became paler until it became yellowish and transparent in the pulmonary arteries. The visceral pleuræ were much swollen, especially where the adhesions with the costal had been, which represented the most consolidated portions of the lungs; here the surface was very ragged and the larger vessels deeply engorged.

The lungs presented every phase of pneumonia seen in swine plague.

Some parts, of recent date, were marked by the engorged condition of the interlobular vessels, in which the blood was coagulated, while the corresponding parenchymatous portions were hyperæmic and at the same time œdematous; the general pale red of the parenchyma was relieved by numerous minute, submiliary centers, which were anæmic and of a greyish, dry appearance; the same invariably embraced the cut section of a small bronchiolus. In other parts of the diseased portions of the lungs the lobuli were of a diffuse, dark red color, but even here the entrance of the air tubes was marked by reddish-grey, more anæmic spots, which were dry and caseous; the tubes were filled with the same kind of material; these portions were also œdematous.

In other parts of the consolidated portions of the lungs the caseous centers were still more extensive and marked by the dull, dry, greyish-yellow appearance; the remaining portions of such lobuli were dark red and œdematous. In still others the entire lobulus presented a dry, yellowish-grey appearance, the bronchial tube being only visible by its more white and refracting appearance; the smaller tubes were compressed, while the larger were filled with the same dry caseous material. In others, again, necrosis had already begun, or the entire parenchyma had been broken down and its place taken by a purulent, aqueous material; the limits of these cavities were marked by the interstitial confines of the lobuli, but in no case were there any indications of indurative processes in the interstitial tissue. In other cases the latter tissue had become complicated in the general destruction, and whole sections of the lung formed quite extensive cavities, filled with the above mentioned purulent material of a decidedly watery character; here the interstitial tissue was much swollen and contained a similar material; where one cut perpendicular to these interlobular spaces, and met them properly, the large engorged veins filled with coagulated blood could be seen as red cords running through them. These cavities had not the irregular shape and ragged base with corroded vessels so often met with in similar conditions in the caseous pneumonia of man; their contents also were more œdematous than puriform in comparison to the latter, and the base of the cavities was smooth, being limited by the interlob-

ular tissue; there is nothing of a chronic character in the above lesions. Cover-glass specimens made from the coagulum in the vessels and the material filling these cavities gave the characteristic bacteria. These pneumonic centers most frequently corresponded to the territory of a single bronchus. These were more lobular as a total consolidation, while essentially lobular from a more analytical point of view; the pneumonia itself was decidedly bronchial in its character, in its initiative stages.

The fauces and larynx were much swollen, their superficial blood-vessels and larger capillaries being engorged; the follicles of the tonsils were filled with a dry, caseous material, while deep-seated ulcerations were also present, covered with a dry, yellowish-gray material, which on removal exposed an eroded ulcerative surface to the view.

The mucosa of the trachea and bronchial tubes was much swollen and the seat of numerous diffuse hemorrhages, as well as those of a smaller and more petechial character. The lumina of the air tubes was filled with a yellowish-red, frothy material.

AUTOPSY IV.

The notes of this autopsy do not give enough variation from the above to warrant entering upon it in detail. There were no ulcerative conditions in the intestines, however. This hog was, on the other hand, a very interesting specimen, being pregnant, and on account of the fact that the characteristic bacteria were found plentifully represented in the blood of a foetus taken to the laboratory and that pure cultures were derived from the spleen as well as the blood.

AUTOPSY V.

Essentially the same. No ulcerations in the intestines. Pulmonary complications very severe.

AUTOPSY VI.

Same. No ulcerations in intestines. Excessive diphtheritic ulcerations in the mucosa of the fauces and larynx.

AUTOPSY VII.

All the above cases, with the exception of No. 1, were from the same outbreak, but this was one of the very few in which excessive choleroïd phenomena were seen before death, hence we only desire

to call attention to the almost total absence of ulcerative, or the so frequently met with circumscribed, indurative conditions in the large intestines which have mistakenly been supposed to have connection with the choleroïd symptoms in this disease. In fact, as in this case, so in nearly all cases where the choleroïd phenomena are excessive, the mucosa of the large intestines will be found of a leaden-gray color, and all acute inflammatory indications will be generally absent. Here we had, indeed, on large, round, cicatricial neoplasmata, most beautifully marked in the colon; it was of a general yellowish-gray color, the circular layers were peculiarly clear, beginning with the smaller central one surrounding an infundibuliform opening; separating it from the surrounding mucosa, which rose somewhat around it, was a clearly defined furrow. See Plate X. The lungs and other organs offered nothing worthy of note from the previous autopsies.

AUTOPSY VIII.

Large black and white hog, with severe choleroïd symptoms, was purposely killed in order to observe the lesions in the intestines in such a case.

Skin but slightly discolored with a few small, diffusely terminating, dark blue spots in the abdominal region. Blood of dark, bluish-red color. About two quarts of a straw-colored fluid in the abdominal cavity. Large intestines agglutinated by a mass of viscid flocculent material; large vessels in the serosa as well as meso-colon and mesentery engorged with blood.

Lymph-glands as usual.

The entire intestinal mucosa, with the exception of that of the duodenum—which was stained yellow—was of a dull, leaden-gray color, somewhat swollen. Not an ulceration, not a button-like neoplasm in the entire course of the large intestines.

I desire to call especial attention to the last statement. It is especially valuable as evidence against the latest misstatement of Mr. Salmon, Chief of the Bureau of Animal Industry, Report 1886—that hog cholera and swine plague are two distinct diseases. It is again valuable evidence in support of my own conclusion, gained by many practical observations in the field, that the ulcerative and neoplastic conditions, which some authors (not observers) have considered so essential to this disease, have no necessary connection with choleroïd

symptoms during life, or with fluid contents in the intestines found at post-mortem examinations. On the contrary, where these ulcerated and neoplastic conditions are the most marked and severe, the animals will be noted to have been excessively constipated during life, except, perhaps, during the last day or two, when septicæmic diarrhœa may set in as the concluding act in the play.

Liver and spleen as usual.

Lungs as severely diseased as in the previous cases.

Diphtheritic ulcerations in the fauces and larynx. Pericardium the seat of ecchymotic hemorrhages in both folds; its sack filled with a yellow, gelatino-fibrinous material, about one-half an inch in thickness, which was united to both the folds of the membrane. Myocardium opaque, yellowish-gray-red, and anæmic to an excessive degree; friable.

Kidneys as usual, with the exception of the pelvic cavity, which, in this case, again, was filled with coagulated blood, the mass being attached to the mucosa. In this coagulum, as well as in the effusions and in the blood, were found many bacteria of the usual kind.

AUTOPSY IX.

The following necroscopical record will be found not only the most interesting one among this series of selected autopsies, but also one of the most important on account of the absolutely exact correspondence between the lesions in the large intestines in this hog and those described by Roloff as occurring in a swine disease in Germany, to which he gave the name of "Käzige Darm-Entzündung."

Middle-sized black and white hog. No discoloration or swelling of the cutis or subcutaneous tissues. Lymph-glands all excessively swollen, presenting, in some cases, a marbled appearance, while others were of a diffuse, dark, blue-red color; in the first, the hemorrhages were in the subcapsular and interlobular connective tissue, while in the latter the parenchymatous substance had been also invaded by the blood. Blood of a dark, blue-red color.

Abdominal Cavity:

About a quart of red-colored fluid in the peritoneal cavity, which contained much flocculent material. The peritoneal lining of the abdominal cavity excessively swollen, and of a clouded-gray color, which was very much interrupted in its diffuseness by an immense

number of petechial and ecchymotic, with occasional diffuse, hemorrhagic centers of greater dimensions. The peritoneal duplicature covering the large intestine was also much swollen and clouded, and covered with a thick mass of viscid material, in which many flocculi were to be seen. The mesenterium was in the same condition, its vessels being engorged with blood. The large intestine was loosely attached to the abdominal wall at three different points. Upon separating these attachments, one could see that there was a circumscribed thickening of the walls of the intestine corresponding to the portion that had been attached; these places were round, about the size of a silver half dollar; the tissues immediately around them were still more swollen than those of the attachments themselves, forming a sort of umbus, or ring, of hard, dense tissue; the serosa in these parts still retained its normal luster, though swollen and clouded. Corresponding to the points of attachment, the surface of the large intestine was granulous, non-lustrous, its vessels injected, and from some there oozed more or less blood from the ruptured points. While the corresponding portions of the costal peritoneum were swollen and clouded, it did not present the same granulous and vascular appearance as that of the intestine at these points; in fact, everything resembling the normal peritoneum was wanting at these circumscribed and indurated portions of the large intestine.

The outside of the small intestine was also of a dull, greyish color. I desire to call especial attention to this fact, for, in this case, the animal was not killed but died in the course of the disease, and yet, as can be seen, and will be still more apparent when we come to the description of the internal conditions of the intestine, there is not a single indication of an acute inflammatory disease along the whole line of the intestinal tract, though the lesions here described must and will completely fill Mr. Salmon's picture of his "hog cholera," described in his report of 1886, but in this, as in every other case, Mr. Salmon's specific "hog cholera microbe" was missed, and "it ever will be missed" in the American swine plague, no matter who seeks it, or how much time they may spend in the hunt.

Stomach: The conditions of the peritoneal covering corresponded to those already described. It was about half full of dark, greenish-gray, semi-fluid mass. Mucosa intensely swollen, forming very prominent rugæ along its base, where it was of a diffuse, deep red color, disturbed

by numerous hemorrhagic centers; the entire mucosa was covered with a thick coating of a viscid material; towards the pylorus this membrane and the covering already described became of a bright yellow, which continued into the duodenum; the mucosa gradually assumed a dull, leaden-gray color, as the jejunum was approached, which extended into and through the ileum. Contents fluid, and of a dirty, olive-green color.

Large Intestine: Contents as above. Mucosa somewhat swollen, and of the same dull, leaden-gray color, and covered with a coating of a viscid character.

We now come to the interesting and important lesions to which attention has been previously called on account of their exact correspondence to those described by Roloff. See Plates IV. and V. Ileo-cæcal valve intensely swollen, indurated, and extending into the lumen of the intestine as a dense, cylindrical body, over one and a half inches in length and over one inch in transverse diameter. The sides and base of this enlarged valve, as well as for about two inches in all embracing directions, presented a peculiar, coal-black appearance, looking as if some one had sprinkled charcoal dust in a thick layer on these parts; the same was not smooth, but had a rough, granulated appearance; the tissues here were indurated to a corresponding degree to those of the valve itself, being at least one-quarter of an inch above the general level of the surrounding mucosa; the whole formed a dense mass of indurated tissue extending to the serosa, which was swollen, non-lustrous, clouded, and very vascular. On cutting through this portion of the wall, one first saw this dense black layer, which gradually assumed a grayish-black color, and then a yellowish hue, until the outer muscular layer was approached, when it became of the color of cicatricial and indurated tissue. Walley makes the singular assertion that this peculiar black appearance here described is due to coal dust, but the fact that coal in any form is a very expensive article in Nebraska, and that it is absolutely impossible for our hogs to come in contact with it, completely answers that argument. On the other hand, there is no question that the conditions above described were anticipated by severe hemorrhagic lesions before the indurative processes began, and that the dark coloring on the surface is due to the action of the sulphureted hydrogen in the intestines upon the iron in the extravasated blood, and not to coal dust.

The free and exposed end of the ileo-cæcal valve presented a very peculiar appearance; at its circumference the black color of its sides terminated, and was replaced by the dirty yellow color of the free end; the walls of this end portion were intensely swollen and roundish; in the middle was the lumen of the intestine, which had thus a very marked infundibuliform character. It was only with the greatest difficulty that a sharpened pencil could be pushed through into the ileum, and as to expansion or contraction, they were entirely out of consideration, so that it would seem a conundrum how material could pass from the small to the large intestine through a passage in such a condition; however, absolutely closed it was not. (Compare this description and Plate IV. with Plate VI.)

Farther down the large intestine, about four inches from the ileo-cæcal valve, were four large, round, sharply circumscribed indurations — the so-called “button neoplasms;” each of these objects was isolated and surrounded by comparatively normal tissues, between which, and these neoplastic productions, was a wall, or ring, of swollen indurated tissue, inside of which was a delicate, yet well marked furrow, on the inside limits of which, again, was the first ring of the neoplastic induration proper; these objects, when in this condition, that of complete induration, are formed of concentric layers of tissues, bearing more resemblance to the gentle wavelets which follow the dropping of a small stone into the water than anything I can describe; the place where the stone sank marking the centre, which is of an infundibuliform nature. This centre was of a dense black color, while the rings surrounding it each became lighter and more yellowish-grey as the peripheries were approached. Like the mass of induration surrounding the valve, and those to be immediately described, these isolated indurations were elevated about one-quarter of an inch above the surrounding mucosa. In the immediate vicinity of these isolated indurations, was a large patch, having the same general characteristics, which was composed of similar productions, which were all more or less in apposition with one another. A large number of similar indurations, but varying much in dimensions, were scattered along the remaining portion of the larger intestine, even to the anterior portion of the rectum.

Liver and spleen as usual.

Kidneys swollen, anæmic, opaque in cortex, and red and injected in

the medullary portion. The outside of these organs, as well as the cut surface of the cortical portion, presented innumerable dark red spots of the size of a pin's head, but every one was of an irregular shape and terminating diffusely: when seen in the cut surface of the cortex some of them were much longer than wide, and their irregular form and diffuse termination was still more manifest.

Thoracic Cavity:

Both sides filled with a reddish-yellow fluid, in which floated masses of honey-colored, gelatinous material. Lungs adherent to the costal-pleuræ, both sides. When removed the surface of the latter was ragged, granulous, the blood-vessels being engorged; from those that were ruptured in the separation of the attached lung there oozed forth more or less blood. The corresponding pulmonary pleura also presented a rough and very vascular appearance, and was much thickened. The pericardial sack was somewhat distended, and contained about two table-spoonfuls of a red-colored fluid. Bronchial lymph-glands as usual. The anterior and middle portions of both lungs were solidified, and presented all the phases of caseous-broncho-pneumonia to the destructive conditions, already described. The entire diseased portions of lungs were œdematous.

AUTOPSY X.

The general conditions were the same as those previously described, with the exception that there were very severe diphtheritic ulcerations in the fauces and larynx. The organs of the thoracic cavity were the seat of very extensive complications, especially the lungs, which were almost completely solidified, but the necrotic disturbances were much more severe than in most cases, complicating a large number of lobuli, and thus forming very extensive cavities, filled with an aqueous, purulent material. The other organs presented the parenchymatous changes already noted.

There was only one neoplastic disturbance in the entire extent of the large intestine; it represented a more acute nature in the process of development than those described in the last autopsy, there having been much more rapid proliferation of epithelium and the formation of a caseous crust, as well as some hemorrhage. This neoplasm was of the size of a silver quarter; it had not any of the peculiar radial appearance of the more slowly developed neoplasms, but was covered

with a dense, black, escharotic mass of friable material, which was in a process of exfoliation, and but partially attached to the underlying tissues. On cutting through this black mass, it was found, internally, to be composed of a grayish-yellow, dry, caseous material; the underlying tissues were but slightly indurated.

AUTOPSIES XI. and XII.

These two necroscopical examinations happened to both fall on the same day, and occurred in the same lot of hogs. They are especially interesting, and equally instructive, on account of the lesions found in the small intestine, also of parasitic origin, but due to the presence of *echinorhynchus gigas* in these cases.

Otherwise the principal lesions in these two hogs did not offer sufficient points of difference from those already mentioned to necessitate going into any exhaustive and finely drawn details. In both there were extensive diphtheritic lesions of the fauces and larynx; the lungs were severely complicated with caseous, broncho-pneumonic, and destructive conditions, the diseased portions being highly œdematous. One animal had a purulent, hemorrhagic pleuritis. Lymph-glands, liver, and spleen as usual. In neither animal were there any indications of ulcerative or neoplastic conditions in the large intestines. On the contrary, in both of these animals, the outside of the small intestine was marked by the presence of quite a number of irregularly formed, bluish-black spots, which were indurated and hard to the touch, and in several of them the wall of the intestine was perforated by a small round opening. There was no hemorrhagic halo around these indurations, nor were the vessels in the serosa injected. These portions of the intestines were ligated and taken carefully to the laboratory for further examination. The contents were semi-fluid, and of a yellowish-green color; the mucosa swollen; in one it was of a dull leaden color, and in the other of a bright yellow shade. The previously mentioned indurations manifested themselves, by internal examination of the intestine, as smooth, sharply circumscribed elevations, about one-sixteenth of an inch in height; they were smooth on the surface and of a whitish color in their peripheries, while towards the center they became yellowish, and then bluish-black; the center was marked by an infundibuliform excavation, in which was attached the proboscis of an *echinorhynchus gigas*, in various sizes and

stages of development. In the intestine permeated by gall the parasites were dead and shrunken, and also stained yellow; while in the other they were dead, but still white and plump. These infundibuliform openings undoubtedly corresponded to the ostia of the solitary follicles, or crypts. In some cases no parasite was attached; the latter varied in length from an embryo one-fourth of an inch long to worms twelve to fourteen inches long. The mucosa immediately surrounding these openings was indurated and swollen in a sharply circumscribed manner, in some cases, while in others there was considerable hemorrhagic infiltration. In others, again, which were about the size of a ten-cent piece, and where no parasite was attached, one observed a yellowish umbus, surrounding a black, raised mass of about the size of a pea, which were frequently in a process of exfoliation. A small pit or indentation frequently marked the center of this black, escharotic material. On cutting across it, it was found to consist, internally, of a yellowish, dry, caseous material, bearing the closest resemblance to similar appearing masses already described in connection with some of the lesions in large intestines.

Without question, the lesions above described have no relation to or connection with the swine plague. They are simply the result of the mechanical irritation of the echinorynchi where the latter have attached themselves to the walls of the intestines, the ostia of the glands offering them the easiest and most acceptable points. But if they have no connection with the swine plague, still, I think, the character and genesis of the lesions serve to throw some considerable light on the genesis of the follicular lesions in the large intestines. In both cases the lesions produced are the result of prolonged and mechanical irritation.

In this case the irritant is an animal parasite, while in the swine plague the irritant causing the lesions is a vegetable organism of much more minute dimensions. These remarks have relation to the follicular, ulcerative indurations and not to the seriated neoplastic productions in the large intestines.

I have no hesitancy in asserting that all the lesions in the large intestines in swine plague are not due to the intra-follicular penetration and action of the germs, but that a certain portion of them have quite another genesis, being due to intra-vascular plugging by the microbes, of which subject I shall treat in another place.

To my mind there is scarcely a doubt that, should the animal parasites be absent and the lesions still remain, in such cases as the above, and a number of hogs perish from pneumonia within a very short period, in a given herd, and, as might occur, the last stages of the disease be accompanied by diarrhœa, that the uninitiated observer would look upon such an outbreak as swine plague, especially if he had heard or read that the peculiar lesions described as occurring in the large intestine are by no means a constant phenomenon in swine plague, as every competent investigator knows.

It is worthy of mention, on account of the so-called "worm theory," which quacks and frauds have impressed upon the minds of our farmers as the cause of swine plague, that these autopsies 11 and 12 were the only two in which I found these parasites in one hundred autopsies.

AUTOPSY XIII.

This case comes from an outbreak of swine plague in an entirely different part of Nebraska, and is especially valuable on account of the history connected with it.

On Dec. 23, 1886, a paper was handed me in which it was mentioned that "a new disease" had broken out among the hogs at Valparaiso, and "that it was not hog cholera, because the hogs bled at the nose." I will here mention what will be considered in detail in another place, that, so far as my experiences have gone, that wherever hogs are dying in numbers, I have never found but one disease, and that disease has been the hog cholera or swine plague, and that, at present, we know of but one disease of the plague kind attacking our swine, and that that is this disease and no other, all and every assertion to the contrary.

The day in question was bitter cold, and the wind blowing as it only can blow here in the West, so that the pleasures of autopsy making in an open field with the thermometer at zero can be better imagined than described, but this is the kind of "field-work" we have largely to do in Nebraska, or else it approaches a torrid temperature.

The owner of these hogs reported that they were all shoats four or five months old, and that they had been selected by himself in person with every care to prevent swine plague. He had purchased them from different farmers, neither of whom had ever had the disease on

his place. His reason for buying them was that he had more corn than he could use, so he rented a vacant lot near his store, so the care of the hogs amounted to little or nothing. Upon this vacant lot was an old barn, the floor of which was thickly covered with old dry straw and litter. Notwithstanding the precautions used in buying and selecting his hogs, the owner failed to appreciate the fact that a lot of 200 hogs had been kept in the same field and had the run of the same old barn in the fall of 1885, and that nearly every one of that lot of hogs had died of swine plague.

The few remaining hogs of this lot were sold off previous to Dec. 1, 1885, since when not a hog of any kind had been on the field or in the old barn.

Within two weeks from the day the owner placed his young hogs on the land they began to sicken, and on December 23d fifteen had died, and a large number were very sick.

As would be natural under such prevailing cold conditions, the hogs at once sought the shelter of the old barn the moment they were unloaded from the wagons, where they rooted around in the old dry straw and litter (in which the hogs of the previous year had died), and, as the owner said, "kicked up a fearful dust." This old straw and litter offered the very best conditions for the preservation of the germs left there by the sick hogs of the previous year, supporting and protecting germs and preserving the moisture during the summer and from the cold of the winter; so that here we have a fact having all the value of a direct experiment, in which the germs of swine plague retained their vitality and virulence for a period of twelve months.

Again, it has been said that these young hogs "kicked up a fearful dust" when they rushed into the old barn to get out of the cold, and the autopsical notes will show that here we also have as exact and trustworthy a dust-aspiration experiment (in which the germs were suspended) under natural conditions, as the most exacting experimentation could demand. This autopsy also shows, as well as the majority of the others herein given, that aside from the blood and glandular organs the chief seat of secondary organic lesions in swine plague is the lungs, and that intestinal complications are of less importance.

A four months old hog, very sick, very weak, labored abdominal

breathing, was placed at my disposal by the owner, with the remark that "it was the best pig in the lot." No choleroïd phenomena present. The owner remarked that "he had not seen any diarrhœa in any of the others until a day or two before death."

Animal knocked in the head. No discoloration of the skin. Blood of a dark, bluish-red color; coagulated rapidly and became red on exposure to the atmosphere. The superficial inguinal, as well as the other lymph-glands of the body, were intensely swollen, and presented the strawberry-like, mottled, red and white surface, so often described in these notes, due to subcapsular and interstitial hemorrhages; cut surface moist and glistening. Peritoneum somewhat swollen and clouded; that of the large intestines the same; small intestines of a diffuse, pink-red color. Mesenterium swollen, clouded, vessels engorged. Liver somewhat swollen, cut surface of a deep, brown-red color, and rich in blood; acini distended, central and inter-acinous vessels very full of blood; but little fatty degeneration to the parenchyma. Gall-bladder nearly empty.

Spleen enlarged, 16 inches long, 3 wide, and 2 thick at superior end. Kidneys somewhat swollen, capsule non-adherent; cortex opaque, anæmic, yellowish-grey in color, variegated by similar dark red spots to those seen on the surface. Medullary substance injected. The mucosa of the entire intestinal tract was but moderately swollen, of a diffuse, pink-red color, varied by a yellowish tinge in the duodenum and jejunum. Valvula Bauhini but little swollen.

Not a sign of ulceration or neoplastic induration in the large intestines.

Thoracic Cavity:

No effusion. Slight double-sided pleuritis, with numerous small points of attachment between the pulmonary and costal pleuræ. Circumscribed, lobular centers of consolidation in the anterior and middle lobes of the lungs. The outer surface of the lungs, corresponding to these consolidated portions, presented a peculiar marbled appearance, but the interstitial tissues in this case were the darker portions, while the parenchymatous were the anæmic and lighter, which is exactly contrary to the conditions presented by contagious pleuropneumonia in cattle. Observers who have been utterly ignorant of every element of pathological anatomy, or how to read the lesions of disease correctly, have mistakenly described these conditions in the

pig, in swine plague, as interstitial-pneumonia; whereas, the fact is, that the interstitial tissues in these cases are almost, if not entirely, free from any neoplastic processes, and especially is it so of any tendency to sclerosis; local indurative sclerosis of these tissues does occur where there has been destruction going on, but the animal eventually recovers. These hogs never come to the notice of the investigator, however, but such lungs can be found at the slaughter houses, and, as said, represent recovered cases of swine plague. I have already said that, in contradistinction to contagious pleuro-pneumonia, the interstitial tissues in cases of swine plague presenting this morbid appearance are red, while the parenchyma is more or less anæmic; this red, streaked appearance is due to the engorgement of the large blood-vessels running through the interstitial (interlobular) spaces, which fill up the entire space; the blood in these vessels, in this condition, is invariably coagulated; the anæmic, diffuse, light, pink-red color of the neighboring parenchymatous tissue is due, self-evidently, to the interruption of the circulation in the interstitial veins, whereby the supply of blood is shut off. The delicate and diffuse pink color of the parenchymatous tissue was interrupted by the presence of numerous, sub-miliary, yellowish-grey spots, in the center of which could be distinguished the walls and ostia of the finest bronchioli; these spots were anæmic, dry, and represented the very earliest stages of caseous, broncho-pneumonia, another condition that observers of this country have described as tuberculosis. The diseased portions of the lungs were decidedly œdematous; great quantities of a clear, aqueous fluid oozing out on pressure. Diapedesis, or hemorrhage, had not occurred in this case. Considerable collateral hyperæmia immediately extended from the consolidated into the neighboring pulmonary tissues, but there was no œdema here. The engorgement of the inter-acinous vessels could be distinctly followed into these tissues, but the vessels were less and less prominent, until they could be no more seen.

Heart as usual. Myocardium opaque, anæmic, yellowish-grey-red in color; tissue very friable.

AUTOPSIES XIV AND XV.

The following autopsy notes are selected from two, of a large number, made upon a herd of swine in which hog cholera prevailed, and in which the owner, a physician by the way, was very much puzzled,

on account of the general scarcity of any lesions worthy of notice in every animal that died. The septicæmia could not be better marked.

First, in no single case did one of the animals that died in this herd of swine present even an intimation of choleroïd symptoms. Above it has been said that the owner was a physician; it should have been added thereto, that he is a very finely educated German physician, and not a practitioner, well to do in the world, and kept a careful watch over his hogs.

I will not go into the details of these autopsies, but simply give the points necessary.

In one of the hogs, the lungs were absolutely clear of any lesions, while in the intestines there were but two small neoplasms about the size of a large pea, which were covered with a yellowish-grey, dry, caseous material. Liver a little swollen, anæmic, fatty. Spleen swollen. Kidneys as usual. Lymph-glands somewhat swollen, and the seat of the usual hemorrhages, but not to an excessive degree.

In the second hog, there were two small centers of caseous consolidation, very dry, and not surrounded by any hyperæmic tissue in the lower part of the middle lobe of the left lung.

Intestines free from any ulcerations.

Other organs as above, except that the kidneys present the most wonderful number of petechial hemorrhagic spots that I have ever seen.

These two cases are especially valuable, showing the true nature of swine plague. The real cause of death was the action of a poisonous principle secreted by the germs upon the centers of respiration and circulation. Again, the bacteria which developed from the splenic cultures of these two pigs were much more malignant in their action, when inoculated, than many other cultures derived from hogs in which the secondary lesions, pulmonary and enteric, were very severe.

AUTOPSY XVII.

Two-thirds grown black and white pig. No cutaneous discoloration. Abdominal cavity contained about three quarts of a light, reddish-yellow fluid. The lymph-glands of the entire body swollen and variegated by hemorrhages and engorged vessels, as has been so frequently mentioned. Large intestines covered over and agglutinated together by a viscid material which contained many flocculi. The external surface of the cæcum and colon was marked by numerous

round, circumscribed indurations, the serosa at these points being somewhat swollen and clouded, and the vessels engorged, while the other parts of the membrane did not show these changes. Small intestine of a diffuse, delicate, pink-grey color. Stomach partially filled with digesta; mucosa swollen and covered with a thick, viscid coating; a few cecymotic spots in its substance. Mucosa of small intestine but slightly swollen, in general anæmic and of a leaden-grey color. Same of the large intestine.

Not a sign of acute intestinal catarrh, not an acute inflammatory phenomenon in the entire intestinal tract, and yet this hog presented severe choleric symptoms for a few hours before it died, but had previously been severely constipated.

Nevertheless, the valve was swollen, and there were several small yellowish patches, of a dry caseous character, but the tissues underneath presented no signs of recent ulcerations; their general level was beneath that of the surrounding mucosa, which rose up wall-like around these caseous centers. At intervals were distributed through the mucosa of the cæcum and colon twenty-four of these neoplastic, circumscribed indurations, with a well-marked center and concentric radiated structure, which have been called characteristic to this disease; these neoplasms varied in size from that of a quarter of a dollar to a silver five-cent piece. They were not covered with any caseous material, nor were they surrounded by any hyperæmic tissues. Spleen somewhat swollen. Liver swollen and fatty. Kidneys swollen, opaque, anæmic, yellowish-grey in cortex; medullary substance hyperæmic; the entire cortex dotted by numerous petechial hemorrhagic centers.

Bronchial lymph-glands as others. Heart flabby, anæmic, opaque, friable.

Lungs normal, except two small lobular centers of recent consolidation in the anterior lobes, one in each.

This case is especially valuable as evidence that swine plague is an infectious septicæmia.

Belted germs found in all the tissues. Cultures made. Virulence proved on mice.

AUTOPSY XVIII.

Black pig, three months old. Movements weak. No diarrhœa. Cut its throat.

No discoloration of cutis. Blood not as blue-red as usual. As the throat was cut the pharyngeal lymph-glands were exposed; they were much swollen and contained several caseous centers. It will be noticed that this is the first time I have met with such centers in my examinations of hogs diseased with swine plague, and that the number thus examined covers several hundreds. The other lymph-glands of the body presented the general strawberry-like appearance so frequently recorded. The caseous condition of the pharyngeal glands antedated the illness of this animal with swine plague, and hence had nothing to do with it.

No effusion in abdominal cavity. The peritoneum of the small intestines swollen and of a deep, pink-red color. Contents semi-fluid. Mucosa swollen; reddish-yellow in anterior portion and red in ileum.

Large intestines: contents semi-fluid, greenish-yellow in color, mucosa swollen, red, occasional hemorrhagic centers, marked by numerous follicular erosions covered with a yellow caseous material, which on removal exposed the swollen edges of the openings of the glands.

Liver much swollen, edges rounded; cut surface anæmic and fatty; acini distended; color, grayish-yellow-red. Kidneys as usual.

Lungs sound.

Belted bacteria present.

AUTOPSY XIX.

General lesions same.

Intestines normal, with the exception of congestion and swollen condition of the mucosa, more especially of the small intestine.

Both lungs the seat of extensive caseous, lobular broncho-pneumonia, and adhered to walls of thorax in nearly their whole appositional extension and with the diaphragm.

Belted bacteria present.

AUTOPSY XX.

Fig, same age. This case is worthy of especial attention, as it exemplifies the very severest phenomena possible of an acute septicæmia and the hemorrhagic conditions peculiar to swine plague in such cases, which are rare under natural infection. Animal died shortly before my arrival.

Skin of the abdomen of one diffuse, blue-black-red color, with nu-

merous spots where there was desquamation of the epidermis and a tendency to ulcerative condition. No œdema! Blood very dark, blue-red; coagulated and became bright red on exposure to the air. Subcutaneous fatty tissue of the abdomen and dependent parts completely filled with petechial and diffuse hemorrhagic centers. The abdominal cavity contained about two quarts of a red-colored fluid. The condition presented by the intestines and peritoneum I have never seen before or since. The parietal peritoneum and that covering the small intestines was marked by the presence of innumerable ecchymotic dots, which looked as if a painter had spattered some bright red paint over the parts; these dots were of irregular size and shape, varying from that of a small pin's head to $\frac{1}{8}$ of an inch in diameter (diffuse hemorrhages were wanting); they were situated immediately in the superficial tissues of the serosa and peritoneum. The large intestine presented a similar picture in its anterior parts, with the exception that there was a marked engorgement of the blood-vessels and the hemorrhages were more extensive and diffuse; as one approached the posterior portion of the colon, they became more extensive until the rectum was reached; here the serosa presented one diffuse, black-blue-red color. The surface of the serosa was intact throughout, these hemorrhages being sub-serous in their entire extent along the greater portion of the large intestine; in the anterior portion, as in the serosa of the small intestine, they were more in the tissues of the serosa proper.

The singular phenomenon about this autopsy was, that while the lesions in the intestinal wall were so excessively severe, viewed externally, that internally they were absolutely nil in comparison. The mucosa of the stomach was somewhat swollen, and marked by a few hemorrhages on the crests of the rugæ, but the small intestine was only of a diffuse, pink-red color and free from hemorrhages; even those in the serosa could not be seen from the inside.

The mucosa of the large intestine was intact throughout, carrying its normal luster even in the most severely complicated rectum. Careful removal of the mucosa of the rectum showed the hemorrhagic condition to be in the reticular tissue between the muscularis and serosa, where the vessels were distended, filled with coagula, and in many cases ruptured, so that masses of coagulated blood and infarctions of an extensive character separated the muscularis from the

serosa. Just here let me say, that the coagula in these vessels and extravasated blood were completely filled with the belted germs.

The omentum and serous covering of the stomach were dotted by similar hemorrhagic centers.

The entire lymph-glands of the body, that is, such as one generally notices, from the inguinal to the pharyngeal, were of one diffuse, blue-black-red color, intensely swollen and œdematous.

Mesenterial and meso-colic vessels engorged with blood of a similar dark color.

Liver swollen, very full of blood, and marked by numerous hemorrhagic spots in its substance.

Spleen, much swollen and full of blood.

Kidneys, surprisingly preserved. Cortical substance clouded only, but the medullary was one diffuse, dark, blue-red color, and the vessels could be followed into the cortical in a somewhat engorged condition, but soon could not be seen. Not a hemorrhagic spot or engorged vessel in the outside limits of the cortical substance. Pelvis filled with coagulated blood, which was closely attached to portions of the mucosa; where the latter was exposed to view it was the seat of numerous hemorrhagic centers of a petechial and diffuse character.

Heart, numerous ecchymotic spots in both folds of the pericardium, and about a table-spoon of blood in sack. Same kind of spots in endocardium. Myocardium somewhat anæmic, brown-red in color.

Lungs normal, with the exception of a few congested lobuli, in which there was an occasional hemorrhagic spot of a diffuse nature, and about the size of a pea.

AUTOPSY XXI.

The following autopsy represents one of the most acute and severe cases of swine plague that we have yet seen, as well as presents lesions that have seldom been described, which, in most essentials, differ materially from those generally found in the writings of other observers, still they are very necessary to a completion of the pathological picture by which this porcine pest displays itself.

Full grown, thoroughbred Poland-China sow. These large sows had been removed from the infested pens about a week after the outbreak began, and put in a yard where there had never been any swine plague, and carefully watched. I visited the "College Farm" at

about ten A.M. of the morning of the twenty-first of December, 1887, and on inquiring how the separated hogs were doing, was informed that there was another fine sow which had just been taken, and was so very sick that she could not get up; also, that the evening before, this animal had appeared apparently well, but that early in the morning it was unable to move. On examining the animal this was found to be the case, and it was decided to kill her at once. She was knocked in the head!

On cutting through the skin, which was in no way discolored, the first thing that attracted attention was the unusually thick, dark, blue-red color of the blood, which was of the consistency of tar, and actually refused to flow from the blood-vessels; in this regard it resembled the blood of animals dying from severe cases of anthrax. It should be remarked that the animal was still kicking when the skin was cut through. The same conditions of the blood existed when the larger axillary vessels were sectioned in the separation of the fore-limbs. This blood also coagulated very slowly on exposure to the external atmosphere, and oxidation occurred in the same manner. Such a condition of the blood is very rarely seen in swine plague, and then only in the most ultra-severe and acute cases. No effusion in the abdominal cavity. Costal peritoneum swollen, greyish, and dotted with innumerable petechial hemorrhages, as well as larger ones of more or less diffuse character. Peritoneal covering of small intestine swollen and of a diffuse, dark, blue-red color on the duodenum, while that of the jejunum and ileum was marked by diffuse patches of a similar color, the balance of the serosa being bright red and marked by numerous engorged vessels and hemorrhagic centers; that of the large intestines swollen, greyish, vessels engorged, hemorrhagic centers more profuse and extensive. Within the folds of the meso colon and that portion of mesenterium attached to the large intestines were immense hemorrhagic effusions in a coagulated condition, which separated the folds of the membranes; the blood-vessels of the latter were engorged and of a dark, blue-red color, the lymph-glands were also swollen intensely and the seat of such extensive hemorrhages and engorgement that they were of the same dark, blue-red color, which entirely obstructed any greyish appearance on the part of the swollen parenchyma. All the other lymph-glands of the body, even those of the inter and submaxillary region,

were in the same condition. The spleen was intensely swollen, and of a blue-black-red color externally, variegated by the greyish appearance of the swollen trabeculae. Contents of the same color and very replete in blood, which flowed from the cut surface as a blue-black-red fluid. Length of spleen sixteen inches, three wide at inferior and two at upper end, three inches thick in the middle of the body.

Kidneys: 5 inches long, 2 thick, and 3 wide in the middle. Capsule non-adherent; outside and cut surface of cortical substance swollen, anæmic, opaque, and of a yellowish-gray-red color; the inter-tubular vessels were much injected, thus marking distinctly the outlines of each tubule; on the cortex they appeared as reddish striæ, or dots, according as the section met the vessels. Medullary substance of a dense, bright-red color. Mucosa of the pelvis swollen, and the seat of numerous hemorrhagic centers, while the cavity contained a mass of coagulated blood, which extended into the veins.

Liver: Swollen, and edges rounded, gall-bladder distended and full of a yellowish-green fluid; outer surface presented a variegated appearance there being larger and smaller territories of a yellowish-gray-red appearance, while others were of a dark, blue-red color; same of the cut surface, which was more or less juicy, the interacinous blood-vessels being distinctly marked in many places and full of blood, while the yellowish-red, opaque centers were anæmic and vessels compressed, the acini were much swollen, and where their characteristics were not rendered indistinct by the engorged vessels and hemorrhage, the intra-acinous vessels were so compressed as to be invisible; at certain points engorged gall-capillaries could be seen as delicate yellow lines.

Intestines: The condition of the serosa has been previously mentioned, but it was so strikingly intensive that it will bear repeating again in this place; the duodenum was one diffuse mass of a purple-red color, although the serosa itself still retained its natural luster. These hemorrhages were mostly in the muscularis, where many of the larger vessels were engorged with blood in a coagulated condition. Along the jejunum the hemorrhages occurred in large, diffuse patches of the same color, they became less in the ileum, where the total engorgement of the vessels was most beautifully marked in the bright-red serosa, at the portions not complicated by these hemorrhagic

patches. The serosa covering the stomach was swollen, clouded, and marked by engorged vessels and petechial hemorrhages. On opening the stomach the extreme severity of the internal lesions was at once apparent. The organ was about half full of a dirty, greyish-green material containing fragments of straw and corn. The mucosa was intensely swollen, and the seat of extensive parenchymatous hemorrhages of a diffuse as well as petechial character; extensive patches of a yellowish-grey color and dryish consistency were distributed all through the mucosa, which were firmly attached to the underlying tissues, but on removal left an ulcerated, granulous surface exposed to view. Towards the pylorus the mucosa had a yellow stain. The mucosa of the duodenum was intensely swollen, and covered throughout with the same greyish-yellow masses, with an ulcerated surface underneath; the same of the jejunum, except that the diphtheritic masses were in patches which became less frequent in the ileum; Peyer's plaques and the solitary follicles were muchswollen; numerous hemorrhagic centers were to be seen in the former. Large intestines: mucosa in the same condition; valve also; the rectum was still more severely complicated, the mucosa being dark, blue-red in color, but covered by the same diphtheritic material which extended deeper into the underlying tissues, the muscularis was of the same color, œdematous and hemorrhagic, a reddish, aqueous fluid oozing out of the cut material; large vessels engorged, and many of them filled with coagulated material. Not a circumscribed ulceration, or button-like neoplasm in the entire length of the large intestine!!

The characteristic ovoid, belted germs were found in immense numbers in the blood, a few in the urine, and sections from the organs also gave a plentiful representation. Cultivations gave positive results.

On Dec. 29, 1887, another large sow died, the autopsy of which so closely resembled the above, the course of the disease being equally acute and severe, that there is no need of recording it here.

NECROSCOPICAL NOTES FROM INOCULATED HOGS.

The previously noted autopsies have been carefully selected from records of several hundred made in different parts of Nebraska during the past two years. They have not, however, been selected to

favor any peculiar views of my own, but to show every phase of pathological lesion which occurs in this very complicated pest, and which can best illustrate its true characteristics.

It now remains to append a few autopsies from the records of pigs or hogs that were inoculated with pure cultivations of the germ of this disease, bouillon cultures being invariably used. The reader will understand that by "pure cultures" are meant unquestionable ones, and that the organism therein was the one described in this report as the only cause of swine plague. In each case the material used was subjected to microscopic and culture control. He will also see how completely the results correspond with those previously detailed as occurring under natural infection.

AUTOPSY XXII.

As stated elsewhere, we visited a very severe outbreak of swine plague at Valparaiso, Neb., in December, 1886. From a pig killed there (see autopsy No. 13) we inoculated two flasks containing sterilized-peptonized bouillon, with due precaution, upon the 13th of December, and upon the 27th of the same month inoculated four six-months-old pigs that were raised at the farm, three of which had been subjected to preventive inoculation in a graded but very severe form, while one had received but one mild inoculation. No. 41 died on the next day, Dec. 28, 1886. At the time these pigs were subjected to this test inoculation, the weather was bitter cold. The inoculation was intra-abdominal in number 41.

As said, the weather was extremely cold. The autopsy was made on the 29th, and the cadaver frozen so solid that the organs could only be removed with great difficulty. They were taken to the laboratory and there examined, there being no opportunity for any changes to occur in transit.

The skin of this animal was one mass of diffuse, bluish-red flush along the entire inferior portion of the belly; the locus inoculationis was swollen and œdemato-hemorrhagic, but otherwise there was no œdema of the sub-cutis. The superficial inguinal, intermaxillary, and other lymph-glands were swollen and the seat of much sub-capsular and interstitial hemorrhage, which gave them that characteristic appearance which has been likened to the section of a large strawberry, as mentioned previously.

The abdominal cavity contained a mass of a straw-colored frozen material, which must have been fluid intra-vitam. Peritoneum swollen and of a diffuse, light pink-red color, dotted by numerous ecchymotic spots; that covering the small intestine was of a vermilion-red color, while the serosa of the large intestines was swollen, grey, and clouded, the vessels being engorged. Mesenterium and meso-colon swollen, greyish, clouded; vessels injected. Same of the serous covering of the stomach. The latter was full of ingesta; the mass was stained yellow where it lay in the pyloric portion. The gastric mucosa was but slightly swollen, but of a diffuse redness in the fundus of the organ. Mucosa of duodenum swollen and stained yellow; that of the balance of the small intestines was not much swollen, but had a diffuse, light pink-red color. Ileo-caecal valve not swollen, as well as the mucosa of the large intestine. Contents semi-pultaceous. Liver very little swollen but quite full of blood. Spleen same. Kidneys very full of blood and somewhat clouded; medullary substance of a bright red color.

Thoracic Cavity:

A slight effusion had taken place in the cavity. Pleurae slightly swollen and clouded, and marked by occasional ecchymotic spots. Pericardium the same. Myocardium somewhat anæmic and opaque in appearance. Lungs normal, except along the edges of the anterior lobes and the deeper portion of the middle lobe, where they were slightly hyperæmic, but without any indications of consolidation. The blood in the pulmonary veins and heart was completely filled with the ovoid, belted germs, which showed every indication of vitality when examined in a hanging drop, and although the spleen was scarcely swollen at all, I have never seen it more replete in these organisms. Notwithstanding the fact that the animal had been frozen almost solid, the cultivations made from the organs and blood of this animal developed with their accustomed rapidity, and proved to be virulent when tested upon small animals.

This case is especially valuable, the animals having neither the pneumonia or intestinal lesions, one or the other of which, or both, most generally occur in this disease. It serves to show that the swine plague is just what we have termed it, *an infectious blood poison*.

AUTOPSY XXIII.

On August 24, 1887, inoculated twenty pigs, three months old, raised on farm and perfectly healthy, with a very weak material. As none of them showed any indication of illness, except a slight swelling and redness on the inside of the thigh where they were inoculated, having for ten days eaten as well as if nothing had occurred, it was determined to sacrifice one of them in order to see if there had been any internal effects. To all outward appearance the animal was in perfect health. Subcutaneous lymph-glands but slightly swollen, somewhat oedematous and lustrous when cut across. No effusion in abdominal cavity, but the folds of the large intestine were covered and agglutinated together by a viscid material, in which some floeculi were to be seen. The serosa covering both large and small intestines was marked by many engorged vessels; the same condition prevailed in the mesenterium. The mesenterial lymph-glands were, however, much swollen, and presented the variegated red and white appearance so often alluded to, though not in an excessive degree; on cross-section, the same presented a similar appearance, there being sub-capsular and interstitial hemorrhage; the parenchyma was swollen, clouded, and oedematous. The serosa of the large intestines presented three circumscribed areas, in the vicinity of the entrance of the ileum into the cæcum, where it was swollen and clouded and the vessels more engorged than over the general surface. On opening the cæcum three fresh ulcerations of the mucosa, with swollen, indurated edges, and covered with a semi-dry, caseoid material were to be seen, corresponding in location to the areas previously mentioned as noticed in the serosa. This caseoid coating was easily removed, the tissues underneath being swollen and of a delicate pink color, with a few very delicate vessels in an engorged condition. The balance of the mucosa of the cæcum and colon was swollen, follicles quite marked, especially those upon the surface of the valve. Mucosa of stomach and small intestines slightly swollen.

Liver swollen, edges rounded, anæmic, aini distended, and of a grey-yellowish-red color. Kidneys swollen, cortex, opaque, anæmic, and of a reddish-grey color, medullary substance hyperæmic. Spleen swollen. Lungs normal! Even from this animal we were enabled to make cultures of the bacterium from the spleen.

AUTOPSY XXIV.

Pig four months old, and perfectly healthy, was inoculated in the abdominal cavity on September 28, 1886; receiving 1^{ccm} of a bouillon culture, it being the third generation of cultures obtained from autopsies made at Rising, Nebraska. On the first of October the animal displayed the first indications of general constitutional disturbance; temperature 104° C., where it remained until the 7th inst., when the pig showed visible signs of emaciation, and had a distressing cough, with a yellowish discharge from the nostrils and a pultaceous one from the rectum. It was killed on the afternoon of the 7th of October, as it was very probable that it would not live until morning.

Obduction: No staining of the cutis. On cutting through the same, the blood, which flowed from the sectioned vessels, was of a dark, bluish-red color, but soon coagulated and oxidized on contact with the air. The folds of the large intestines were agglutinated by a thick, viscid material, in which were numerous flocculi. Superficial inguinal, mesenteric, and other lymph-glands were much swollen, the cut surface being moist and glistening, and of a diffuse, red color, interrupted by small interstitial and sub-capsular hemorrhages. Mesenteric vessels engorged, and of a dark, blue-red color. The serosa covering the small intestines was of a diffuse, light, pink-red color; on opening these the mucosa was found swollen, with diffuse capillary redness; Peyer's plaques swollen, with occasional hemorrhagic centers within them. Ileo-cæcal valve intensely swollen. Throughout the large intestines were numerous ulcerations, varying in size from that of a pea to a ten-cent piece; they were covered with a greyish-yellow coating of a friable character, which on removal displayed a reddened surface, the edges of the ulcerations being irregular and tumefied, while the surrounding tissues were somewhat hyperæmic. Button-like neoplasms were entirely wanting.

Spleen swollen; its parenchyma was of a dark, blue-red color, and very rich in blood. Liver swollen, edges rounded; acini distended, cut surface of a greyish-yellow-red color, opaque, and anæmic; interstitial tissue marked by engorged condition of the bile ducts. Kidneys: capsule non-adherent; cortical substance anæmic, opaque, and of a yellowish-grey-red color; medullary substance of a bright, pink-red color.

Pleural Cavity :

Slight effusion of a red-yellow color. Pleura swollen and dotted by numerous hemorrhagic centers of a diffuse and petechial character. Pericardium in a similar condition ; its sack contained about a table-spoonful of a reddish-yellow fluid. Endocardium also swollen and dotted with numerous ecchymotic spots. The anterior and middle lobes of lungs, as well as a portion of the posterior lobes, were consolidated or hemorrhagic, the complications being essentially of a lobular character, being sharply outlined from each other by the engorged condition of the interlobular vessels ; the lobuli themselves presented every phase of pneumonia, from that of engouement with a diffuse, dark red color, to a complete cellular infiltration, in which case the lobuli were anæmic and of a yellowish-grey color ; in all cases the complicated portions of the lungs were œdematous ; the termination of the engorged inter-acinous vessels marked also the extent of the œdematous tissues.

Tracheal and bronchial mucosa swollen, the tubes being filled with a foamy, straw-colored material, which in the deeper seated ones became more fluid.

Belted, ovoid germs in all materials examined, as well as successfully cultivated and inoculated in mice.

The same general results occurred in pigs number 17 and 18 which were inoculated with the same material, as well as with numbers 6, 7, and 8, which were inoculated with pure cultures from the first case of swine plague from which we obtained material in July, 1886, and pig number 14, which was inoculated with the tenth generation from that material.

AUTOPSY XXX.

Six months old pig, in fine condition. Inoculated October 27, 1886, with 1^{ccm} of a bouillon culture of the germs of swine plague. On account of the distance of the farm from the laboratory, and then having no conveyance, it was impossible to get any accurate account of the clinical phenomena in most of these cases, as we were entirely dependent upon the feeder for such. In this case, it was simply reported "that the pig had not eaten very well." It died on the seventeenth day of November, or twenty-one days after inoculation, which was rather a protracted period, but the pathological lesions were among the most severe we had yet met with in any of our inoculated

hogs, especially in the intestines, and it is for that reason that the case is introduced here.

Skin normal. Blood of the usual dark red color. Slight effusion in the peritoneal cavity. Peritoneum clouded and swollen. Lymph-glands as usual, but not to an excessive degree. Liver, swollen, anæmic, opaque, yellowish-gray-red in color. Spleen as usual. Kidneys swollen, opaque, anæmic, as usual. Stomach: mucosa excessively swollen and gathered into deep rugæ; very extensive diffuse hemorrhages through the substance of the membrane; towards the pylorus it was stained yellow, which color extended through the duodenum; mucosa of the same much swollen; that of jejunum and ileum in the same condition, and of a diffuse, pink-red color; Peyers' plaques and solitary follicles swollen; in former a few small hemorrhagic centers. Large intestines: Mucosa swollen, and of a leaden-gray color, with some centers of diffuse hemorrhage; ileo-cæcal valve intensely swollen, indurated, and extended into the intestine for over an inch in length; it was surrounded by an irregularly shaped mass of a dirty, dull black color, the surface of which was granulous; the same material extended up the sides of the indurated valve, until it reached the crown, which was of a dirty yellow color. (See Plate VI.) This mass was about one-quarter of an inch in thickness; underneath the black surface it was of a yellowish-grey color and friable consistency, while still deeper the tissues were indurated and of a more whitish-yellow color; the tissues adjoining were also much swollen, with a few ecchymotic spots scattered through the mucosa. All through the cæcum and colon were numerous small follicular ulcerations, covered with a dryish, yellow, friable mass, the edges of the same being indurated. Button-like indurations wanting.

The thoracic cavity contained about a pint of straw-colored fluid. Pleuræ swollen, clouded, general color greyish interrupted by numerous ecchymotic hemorrhages. Pericardium in the same condition; its sack held about a table-spoon of the same colored fluid as was met with in the pleural sacks. Myocardium anæmic, opaque, swollen, and yellowish-grey-red in color. Endocardium swollen and clouded; dotted with ecchymotic spots. The pulmonary pleuræ were attached to the inside of the thoracic cavity in several places. Cross-section of the lungs revealed marked lobular pneumonia in the anterior and middle lobes, and some in the inferior; the same were in all condi-

tions common to this disease, from simple hemorrhagic conditions to the infiltration and destruction of the tissues of the lobuli; the interlobular vessels were distended by a bluish-red coagulated material; the lobuli in the immediate vicinity were heavily engorged with blood, but those at a distance were uncomplicated. Œdema as usual. Mucosa of the trachea and bronchial tubes swollen; vessels injected, with petechial and small, diffuse hemorrhages scattered here and there.

This animal was also frozen hard before the autopsy was made, but allowed to thaw out in the laboratory. The spleen was at once removed, and agar-agar cultures made therefrom, which developed as usual. Positive results from same on mice, the same ovoid, belted germs being present in their blood.

(This was certainly a case in which, according to Mr. Salmon, his imaginary germ of "hog cholera" should also have been found.)

AUTOPSY XXXI.

This hog has quite an interesting history in connection with our experiments. It belongs to the group 41, 42, 43, 44, 45, 46, 47, experimented with in 1886, and is recorded as "No. 45." This animal's part in the programme is as follows: Nov. 4, 1886, inoculated, with others, with a weak vaccine from which no visible disturbances were to be seen. Nov. 27, '86, re-inoculated with a stronger virus.

Dec. 26, '86, a test inoculation was made upon these hogs with a primary bouillon culture made from material from an outbreak of swine plague at Valparaiso, Nebraska, the germs of which were so virulent that test hog No. 41, which had only been inoculated once, succumbed in about twenty-four hours. (See Autopsy XXII.) The other hogs withstood even this test, and were all alive up to Aug. 6, 1887, when Nos. 42, 43, 44, and 45 were again subjected to a test inoculation with a recently prepared and strong virus, each animal receiving six fluid grammes in the abdominal cavity. Of the four hogs thus tested, only this one, No. 45, showed any signs of illness, being off its feed for about ten days, lying continually in the straw, and growing gradually worse, so that I killed it in order to be sure and have the cadaver fresh, as the animal would have probably died during the ensuing night. It must be remarked that during its illness, this animal did not have any choleroïd discharge whatever; in fact, constipation was the marked phenomenon with reference to intestinal action.

This fact deserves the most careful remembrance in connection with Mr. Salmon's assertions that hog cholera is a separate disease from the swine plague, and characterized by a germ, which does not color at its pole ends like the bacterium of rabbit septicæmia. Mr. Salmon's hog cholera is an ulcerative enteritis!

This hog was inoculated with a culture of the germ described in connection with the etiology of this disease, in this report, which does color at its poles, and which has a clear center; and, as the results will most conclusively show, is the true and only germ of hog cholera or swine plague, and which does cause the lesions found in the intestines in this disease, which statement I positively and dogmatically reiterate, irrespective of the false assertions that have emanated from Washington.

No. 45 was, at this time, about sixteen months old, but had only arrived at about two-thirds of the size it should have, on account of the severity of the inoculation tests to which it had been subjected.

No discoloration of the outside cutis observed. Blood of an extremely dark, blue-red color, both arterial and venous; it coagulated slowly upon contact with the air, and finally became bright red in color. Panculus adiposus very white and somewhat atrophied; both it and the sub-cutis were free from any hemorrhagic effusions. Small quantities of a dark, bluish-red fluid oozed from the openings of the segmented blood vessels. Subcutaneous lymph-glands excessively swollen, and presented a mottled appearance, being of a greyish color in some parts and bluish-red in others, owing to subcapsular and interstitial hemorrhages; cut surface œdematous and of a similar appearance.

Abdominal Cavity:

On opening the same the first thing that struck the eye was the pregnant uterus, which contained seven foeti. About a quart of straw-colored fluid containing much flocculent material was in the cavity. On endeavoring to lift up the omentum, it was found to be closely attached to the large intestines in three different places; separation was impossible, so that these spots had to be cut around in order to free the membranes. Costal peritoneum pale and free from hemorrhages, somewhat swollen and clouded. Visceral peritoneum presented the same general characteristics, except at the points where its omental fold had been attached to the cæcum and colon; here it

was much thickened, rough, and very vascular; these points were sharply circumscribed from the balance of the intestinal wall, being raised above the general surface, and were also much more extensive than any such conditions that I had previously met with in the course of my investigations. The large intestine was much contracted, but all along its course could be felt hard lumps of varying sizes, filling the haustra or sacculations. The rectum appeared contracted and empty along its entire course.

The reader will be kind enough to bear in mind that we are describing a case of inoculated swine plague, caused by the belted germ, which colors at its poles, which Mr. Salmon says is the cause of "chronic pneumonia" only!

The small intestine was also contracted, and of the same, anæmic, pale-grey color. Along the course could be felt a number of small, circumscribed indurations, but they differed from those in the large intestine from the fact that the serosa over them had not lost its lustre, and that in the center of many of them the continuity of the wall was interrupted by a small round opening. Stomach: The outside was of the same pale, greyish color, somewhat swollen and clouded; its walls were also contracted; contents, mostly dirt; mucosa swollen, covered with a viscid, adhesive coating; generally anæmic; pyloric portion stained yellow. Small intestines contracted and empty during their entire course; mucosa not much swollen, very anæmic; stained yellow in the duodenum. Quite a number of *echinorynchi*, in various stages of development, attached to the walls were found in the intestine.

Large intestines: Not an indication of acute inflammation! Mucosa anæmic, not swollen, of a delicate, pinkish-yellow color. Very little, if any catarrhal covering. In general, quite empty, but here and there were to be seen large, greenish-black masses of feces in the form of balls, filling the haustra of the colon; the cæcum and rectum were empty. In the immediate vicinity of Bauhinis valve was a dense, black, circumscribed mass, larger than a silver dollar, with a well marked center-piece about the size of an old-fashioned silver three-cent piece; this mass was the thickest at the center, and gradually grew thinner towards its peripheries; its surface was marked by a continuous line of consecutive ring-like elevations, extending from the center-piece to the circumferences; on cutting through it,

the superficial mass was seen to consist of a caseous, dry, friable material, while the underlying tissues were nothing but a mass of dense, indurated, connective material. The mucosa around this object was separated from it by a slight furrow. In the *cul-de-sac* of the cæcum were a number of smaller objects, having the same general characteristics, to which small masses of fæces were attached. Three inches deeper down the cæcum was a similar black patch, composed of two of these large indurations, which complicated the entire intestinal wall in a transverse direction. A special description of the same need not be given. As one approached the colon, still another of the patches was met with, and at the termination of the colon still another, which was two and one-half inches in its longitudinal diameter and one and one-half in the transverse, these measurements bearing relation to the course of the intestine. The surface of this last induration was somewhat smoother than that of the others, the radiations not being well marked. See plates VII. and VIII. Rectum free from ulcerative or indurated conditions. (It does not seem as if any one could have seen more extensive lesions in the large intestine, which Mr. Salmon says are characteristic of his "hog cholera," yet his manufactured germ of "hog cholera" never had any connection with the lesions in this hog.)

Spleen much swollen; 14 inches long and 2 thick at the superior end; surface marked by numerous irregularly-shaped vegetations, pulp, juicy and rather dark in color.

Liver swollen, edges rounded, capsule marked by numerous vegetations; gall-bladder half full of a thick, greenish-yellow material; cut surface anæmic, of a greyish-brown color; opaque but with a slightly yellow tinge. Acini very much distended. Central vessels invisible; the interacinous gall ducts were somewhat injected, to which was due the "yellowish tinge" previously mentioned. Kidneys swollen, capsule free, corticle substance of a dull, greyish-yellow appearance, anæmic, swollen, opaque. Medullary substance of a dark, blue-red color, rendered striated by the still more intensive color of many distended vasa-recti.

Thoracic Cavity:

No effusion! No adhesions! Pleuræ normal! Heart flabby, muscle anæmic, opaque, yellowish-grey-red in color; small quantity of semi-coagulated blood in ventricles.

Inferior portion of the middle lobe of both lungs and a portion of anterior lobes were the seat of much lobular pneumonia in various stages of development, and cedematous.

Lymph-glands of the entire body as described for the subcutaneous. (Mr. Salmon's latest report having just come out at this time, in which he endeavors, but in vain, to save his reputation as a scientific investigator, by reporting the intestinal lesions from swine plague, and thus makes a new disease called "hog cholera," for which he manufactures a nondescript germ, it became a matter of urgent necessity to subject such a case as this to the most rigid examination; for, as has been said, there was no doubt about the presence of the bacterial culture used to inoculate this animal, and that the germs in it were the same we have invariably found in every case of swine plague.)

The blood, blood serum, and all the organs were subjected to the most rigid microscopical examination, and the material selected treated with such care as to render the presence of adventitious germs impossible. Hanging drop cultures were also made direct from the blood serum, as well as others upon different media.

After several hours spent at this useless job, we were unable to find any other micro-organism than that invariably found in every case of swine plague we have investigated, and which has been described as a "belted, ovoid organism which colors at its pole ends," and which bears no resemblance to the Washingtonian-Bureaucratic nondescript.

SWINE PLAGUE IN GREAT BRITAIN AND EUROPE.

SWINE PLAGUE IN GREAT BRITAIN.

The purpose of this report being to show that there is but one swine plague known to exist in the United States, and that this disease is identical with the disease known by the same name in Great Britain, as well as that discovered by Cornil and Chantemesse and Rietsch and his colleagues in France, in the summer of 1887, and the disease in Sweden and Denmark, as well as to show that it exists in Germany, though not yet recognized as an independent malady, the reader must pardon the necessity of making the same as exhaustive as possible, for it is the writer's desire either to settle the question once for all, or place it in such a condition that it must be soon done, in order that the swine growers of this country need not be in further danger of the humbuggery that has emanated from Washington with regard to a second and entirely independent porcine pest, to which the name "hog cholera" has been given. In considering the history of the micro-etiological organism of swine plague, it has been shown that Mr. Klein, of England, saw the same as far back as 1876, and that he gave illustrations which conclusively prove that he did, though he entirely failed to recognize it as an independent species from the one he considered to be the true germ; which he says resembled "*Bacillus subtilis*." It has also been said that the author was the first to discover the germ of English swine plague in the fall of 1886, and that the same is identical with the bacterium which caused the American swine plague, but he distinctly wishes it understood that he places more stress upon the identical nature of the lesions and clinical course of the disease than upon micro-organismal resemblances. This assertion has been again confirmed by the examination of some material sent me by Mr. George A. Banham, a very eminent English veterinarian, living at Cambridge, England; that the material came from an animal that had died from swine plague was evident from the piece of the large intestine sent with it, which contained the so-called "characteristic ulcerations," of varying size, covered with a yellowish-

grey, friable, caseous material. This material was received March 25, 1888, and when subjected to a microscopic examination exactly the same micro-organism was found to be present, and in great numbers, as that found in the specimen of Dr. Bowhill, in 1886. The lungs of this animal, while heavily engorged with blood, contained no visible centers of consolidation, and yet those conditions of the intestines were present which Mr. Salmon claims are the essential characteristics of his manufactured disease, "hog cholera." No such micro-organism as he describes as the cause of his peculiar and imaginative disease could be found in these tissues!

I now desire to call especial attention to Mr. Klein's description of the lesions of swine plague in England, which the reader will have no difficulty in seeing are the exact counterpart of those described in the various autopsies selected from the notes of those made during my investigations in Nebraska.

Mr. Klein says:*

"Intestines: The small intestine shows, as a rule, hyperæmia, and in some cases ecchymosis in the mucous and submucous tissues and the serous covering. In some rare cases the same is also seen in the stomach. The large intestine always shows the most characteristic appearances. There are at least smaller or larger isolated or confluent, generally roundish, ulcerations at and around the ileo-cæcal valve, the rest of the mucous membrane being hyperæmic even to ecchymosis. In the highest degree the whole of the large intestine, down to the rectum, contains ulcers; in the cæcum they are confluent, and measure several inches, extending transversely as well as longitudinally; while the whole remaining mucous membrane of the large intestine is much thickened, and in some parts the submucous tissue contains large accumulations of blood. The ulcers are of various aspects. The following forms may be seen; very minute, well defined, prominent, yellowish-whitespecks, of the size of a millet or hemp seed; then somewhat larger, more flattened, prominent, circular or oval, yellowish patches of the size of a hemp seed, up to about one-eighth of an inch in diameter; next, flat, circular, or slightly oblong patches, situated on the crest of a fold of the mucous membrane, in size from one-eighth to an inch in diameter, generally black, or grey, except a very conspicuous, and, I may almost say, characteristic prominent rim, which is yellow. The ulcer generally shows a pale, central, or eccentric disc, around which the rest of the ulcer is arranged as concentric rings. (See plate.) Between these flat ulcers with concentric layers and those uniform, yellowish-white, prominent patches and nodules, there are all intermediate forms;

*Veterinary Journal, Vol. V., 1877, pp. 45, 123.

this is easily understood if it is borne in mind that as the latter increase in size, the central part is transformed into that black or greyish mass.

"The following lymphatic glands present very characteristic appearances: the mesenteric glands, especially those of the large intestine; the bronchial glands; the chain of glands along the descending aorta; the sternal glands, and the submaxillary glands. The glands are much swollen, slightly firmer than normal, more or less red, in severe cases dark purple or even black, and when cut into a considerable amount of red fluid oozes out. At the same time it may be seen that the chief seat of red colorization is the cortical part of the gland, from which it extends into the medullary part according to the greater or smaller severity of the change.

"In a few cases we observed ulceration of the organs of the throat." * * * *

I now desire to call the especial attention of the reader to the following remarks of Dr. Klein, which will be found to be absolutely identical to the experiences of Dr. Detmers and myself in investigating the swine plague of this country.

Klein says:

"Further important symptoms, and which have not hitherto received due attention, are affections of the lung and serous membranes!! Especially the lung affection has been as constant as any of the preceding, even more constant than that of the skin. The state of the lung is this: the slightest degree consists in a distinct mapping out of the lobes and lobules by œdema of the interlobular tissue, the lung tissue of the corresponding parts being at the same time hyperæmic. Then these parts become hepatized and transformed into a heavy, airless, red, transparent tissue; and subsequently smaller or larger opaque or white specks and patches appear in the red substance, and, as they increase in size, become gradually confluent. On section it may be seen that this is due to the fact that the bronchial tubes become gradually filled up with a white, brittle, cheesy mass, progressing gradually from the finest ramifications on to the larger branches of affected lobules and lobes. Finally, the whole lobule is transformed into a discolored, dry, hard, friable mass. The pleura corresponding to these parts is of course inflamed, being in some cases exceedingly thick and covered with false membranes. In severer cases the greater part of one lung and portions of the other may be thus changed, and, on the external surface there may exist smaller or greater ulcerations. Except in very slight cases there is generally a certain amount of pleural exudation; and in severer cases the pleuræ contain a considerable quantity of a thick, offensive, yellowish, or dis-

colored exudation. In some cases the pericardium is also inflamed and contains a large quantity of exudation, the membrane itself being much thickened. We have had cases where there was no skin eruption, slight intestinal change, and extensive pleuro-pneumonia, pericarditis, and pleuritis." Sic! Salmon.

"The next organ of importance is the spleen, which is of a dark color and sometimes enlarged and at others not. The liver, in severe cases, is enlarged and very full of blood. [Strange that Klein could not see Salmon's specific "Cirrhosis" in one single case!] The kidneys are sometimes [!? always—B] also changed; there is hyperæmia of the pyramidal parts, and underneath the capsule, which may be easily stripped off; there are visible, on the surface of the cortex, small, round, hemorrhagic spots of the size of a pin's head, while similar spots, but scarcer, are met with in the pyramids. The severer the case the more numerous these hemorrhagic spots."

I shall show that these hemorrhages have no connection with the severity of the case, but are probably connected with Mr. Klein's bacillus, which resembles *B. subtilis*, and are an accidental phenomenon.

Klein sums up as follows:

"Thus we may see that the skin, large intestine, lymphatic glands, and lungs are the organs most commonly affected, and that changes of the serous membrane, spleen, liver, and kidneys, and the organs of the throat are less constant."

THE VIEWS OF PROF. WALLEY,*† PRINCIPAL OF THE DICK VETERINARY COLLEGE, EDINBURGH, SCOTLAND, UPON SWINE PLAGUE IN GREAT BRITAIN.

"Synonyms—Hog cholera, typhoid fever, and enteric fever of pigs, blue disease, red soldier, measles, pig distemper, contagious pneumo-enteris.—(Klein).

"As in the case of other diseases so in this, many synonyms have been employed to designate it, some of them referring to some characteristic external symptom, as the color of the skin or the character of the skin eruptions, and others to some fancied resemblance in its internal lesions to those of well-known forms of disease. Unquestionably the present official synonym, 'swine fever,' or better 'swine plague,' is the most simple and the most useful for all practical purposes.

"Definition.—A specific eruptive fever, peculiar to the pig.

"Nature and Characteristics.—Many years ago, when I first be-

* From the Third Annual Report of the National Veterinary Association of Great Britain.

† On account of its important bearing upon this question the essay of Prof. Walley is given in full.

came familiar with this malady, I looked upon it as a sporadic or enzootic affection, due to an altered condition of the blood, as the result of purely local causes, *e.g.*, bad hygiene and improper or impure dieting; I was of opinion, in other words, that, like purpura hemorrhagica, it was simply a blood lesion, and in my earliest lectures on the subject described it as such, and drew parallels between its lesions and those of purpura. Nor did I stand alone in this estimate of its character. In America it was also mainly attributed to bad hygiene and bad dieting; but earlier in the United States than here it was gradually recognized as a zymotic disease and of a contagious character.

"In the United States of America it was taught to be of the nature of cholera [Salmon!] and this is not to be wondered at when we consider its fatality, its rapid spread, the enteric conditions with which it is associated, and the state of collapse into which its victims are sometimes thrown.

"Subsequently an idea prevailed that the disease was allied to the typhoid fever of man, and that, too, mainly from the fact that necrotic enteric changes were common; but as early as 1876 or 1877, Klein showed that a great mistake had been made, pointing out that the lesions of human typhoid were confined mainly to the small intestines, and were localized in the lymph follicles thereof; and further, that the necrosed tissues on desiccation leave behind pit-like depressions, and not unfrequently result in perforation; whereas in swine plague the ulcerative lesions are confined mainly to the large intestines (often grouped around the ileo-cæcal valve), seldom extend to the small intestines, and are still less often found in the stomach. The lesions are not localized in the lymph follicles, do not as a rule extend deeper than the superficial mucosa, and rarely produce perforation. About 1878 Klein suggested that the disease ought to be designated 'contagious pneumo-enteritis,' and he did this on the assumption that pneumonia was a constant lesion; in this, however, he was certainly mistaken, as I have seen numbers of instances in which lung lesions were conspicuous by their absence.

"In 1877 Klein drew attention to the presence constantly, in the local lesions of the disorder, of colonies of micrococci, and to these he was evidently inclined to attribute specific properties.

"In 1878 Klein drew attention to the fact that there existed in the peritoneal effusions and elsewhere a bacterium which, in its morphological characters, bears a close resemblance to the bacillus of hay infusion (*B. subtilis*), and in some respects to bacillus anthracis; the important difference being that the bacillus of swine fever is finer than either of the others, and although multiplying by spores, it is motile and somewhat rounder at its end. Recently Pasteur has described a dumb-bell shaped microbium. Klein looks upon these as foreign to swine fever.

"Not only by Klein, but by Professor Law and others, has the virus of swine fever been cultivated and attenuated, and with the modified virus animals have been inoculated, and, according to Laws, protected; but Klein's experiments in this direction show that even after several inoculations the virus is still capable of producing specific symptoms, though the mortality is materially reduced. Not only has the pig been successfully experimented upon; rabbits and mice are shown to be susceptible to the action of the virus.

"Swine fever is no respecter of persons amongst the pig tribe. It attacks the young, the old, the fat, the lean, the well and ill-bred, the male and the female alike; but in my experience pigs from six to eight weeks to several months old ('shoters' as they are called in Scotland) are most susceptible, and in them the mortality is greatest, while large pigs, as sows, frequently escape it, or if attacked, recover. Young pigs in utero often die and are aborted, or if they survive through the normal period of parturition, are puny and short-lived; [In my researches the reader will find an account of finding the germs of swine plague in the unborn foetus of a sow that had died from the disease.—B.] while sucklings not unfrequently pine away one by one, until the whole litter is exterminated, even before the mother gives any evidence of being herself affected with the disease. Frequently, too, the mother will pass through the nursing stage apparently in good health, but after the offspring have been weaned, or have died out, she will gradually emaciate, and ultimately succumb to enteric, hæmal, or pulmonary disease. There are those who attribute the death of the foetus in utero to the fact of its being supplied with impure blood, or to the histological changes in the placenta, and not to the direct action of the virus of the disease. Such a statement has been made in reference to anthrax, and even some of our best authorities declare that anthrax lesions are never found in the foetus (or its blood) in utero, the placenta acting as a filter and effectually preventing the passage of the bacilli. To this statement I give a flat denial. I have found extravasations and effusions into the tissues of the unborn lamb, and I have found bacilli in these products and in the blood when I have failed to find them in the blood or tissues of the mother. In swine fever, as in other zymotic affections, abortion is often the salvation of the pregnant female.

"Swine fever may assume either an enzootic or an epizootic character. Probably never within the memory of the present generation of veterinary surgeons, in this or in any other country, has it spread so rapidly or so widely as in the prevailing epizootic. What the determining cause of this widespread outbreak will be, it would be difficult to say; but I am satisfied of one thing and that is, that the disease has in the past been practically left to wander as it listed;

that it has existed in certain districts for weeks or months before it has been detected or recognized; while during the whole of that time animals from the infected areas have been sent into surrounding districts, and have thus scattered the disease far and wide. Moreover, a remarkable amount of ignorance as to its diagnosis has been exhibited, even by veterinary surgeons, and it was on this account mainly that I suggested to the provisional committee of the National Veterinary Association that the subject of swine fever would be an appropriate one for discussion at the Birmingham meeting.

"Swine fever, perhaps more than any other similar malady, presents itself under a variety of aspects; in other words, its visible manifestation and the localization of its lesions in different outbreaks assume very different characters. In its clinical character it is hydra-headed, and this fact alone is sufficient to explain the apparent anomaly that scientific men have not always been able to recognize it when the malady has been for the first time brought under their immediate notice. Not only so, but even in one and the same outbreak it often passes through different phases ere it runs its course; thus: the first few animals attacked may show no external manifestations of the disease; they may pine and waste gradually away, perhaps coughing or purging to some extent, but showing no symptoms sufficiently pronounced to raise even a suspicion in the mind of the owner or the attendant as to the nature of the malady which is gradually but surely emptying the pig-sties of their occupants. At another time a number of animals are attacked with symptoms of narcotic poisoning—staggering about their sties, boring their heads against the wall, passing into a state of convulsions, foaming at the mouth, and champing the jaws or gnashing the teeth, as the pig is wont to do when under the influence of a cerebro-spinal excitant. Again, the animals appear to be suffering from rheumatic cramp and when called upon to move not unfrequently do so with their fore legs spasmodically flexed, the motive power being supplied entirely by the hind legs. But after a time, in each of these cases, characteristic enteric, eutaneous, or pulmonary lesions are developed, and the matter is set at rest. In other outbreaks the lungs are most largely the seat of organic changes, while the existence of bowel lesions is the only exception that proves the rule; [Sic! Salmon!] and lastly, there are cases in which the skin and enteric lesions are so pathognomonic as to leave no doubt in the mind of the veterinary surgeon as to the nature of the malady with which he has to deal, and there are others in which no sign (external or internal) exists to show what the disease actually is. [See account of autopsy on experimental pig No. 41, in which there were no characteristic lesions.—B.]

"Swine fever is in every sense both infectious and contagious. It is disseminated (to limited distances) through the medium of the air;

by means of such fomites as water, food, litter, urine, faeces, dung; by other animals, and above all by ducks, poultry, and rats; by contaminated conveyances, such as railway trucks, floats, carts, and vessels; by the dirty boots and clothes of attendants; by the actual cohabitation of healthy with diseased animals, and by direct inoculation. I was at one time inclined to think that in those outbreaks in which lung lesions were most pronounced the disease had been contracted by inhalation, while in those where bowel lesions were most prominent it had been contracted by indigestion, just as I believe is often the case in anthrax; but on considering the matter more closely in the light of our present knowledge of the fact that, as a rule, in most of these maladies the virus must gain access to the blood before it can produce its characteristic effects, I have been led to modify my opinion in this respect.

"Swine fever does not necessarily attack all animals exposed to the influence of the virus—a very large percentage escape—nor is it in every outbreak equally fatal. In years gone by I used to treat this malady with great success, and certainly saved fifty per cent of the animals treated; and even now I feel quite satisfied that numbers of pigs pass through the disease without manifesting any recognizable external symptoms, no matter how carefully they may be watched, and recover.

"Does one attack of the disease protect against future attacks? According to the evidence afforded by Dr. Klein's experiment on the protective value of swine fever virus, the question should be answered in the negative; but in the past few months I have had the opportunity of watching several animals that during a previous epizootic were attacked by this malady and recovered, and which during the present outbreak have successfully resisted its influence.

"Swine fever, so far as I know, has never been transmitted to any other of our domestic animals, nor except experimentally to any other creature, and while mice may be successfully inoculated with the cultivated or natural virus, the rats seem to enjoy perfect immunity from its effects—at least so I conclude from the fact that these animals cohabit with pigs (in all stages of the disease), and live with them on the most intimate terms, without showing any signs that they were in the least degree affected by the virus of the disease. Swine fever is as pre-eminently a pig disease as is cattle plague a bovine disease.

"The extent of vitality possessed by the virus of swine fever has as yet to be determined; but of one thing I am quite assured from experience and observation, and that is, that it may retain its vitality for several weeks, if not for months, in dirt or other suitable pabula.

"Its incubative stage has been shown experimentally to be confined to about four or five days, but naturally it is about ten or twelve days.

"Its duration is somewhat indefinite. As in the case of pleuro-pneumonia, so here, an animal may be laboring under the influence of the virus for many days without attracting the attention of those persons who have charge of it. On the contrary, it may be well, and dead in the course of several hours, or two or three days; while in not a few instances it runs a sub-acute or chronic course, which may extend over several weeks, or a month or two.

"SYMPTOMS AND COURSE.

"The invasion of swine fever is sometimes very pronounced; at others its actual existence is evidenced by indeterminate symptoms, its special characteristics being slowly and insidiously developed.

"CONSTITUTIONAL SYMPTOMS.

"In the earliest stages there is always more or less fever, the temperature being elevated two or three degrees, the pulse increased in rapidity, the bowels constipated or relaxed, occasionally there is vomition, and sometimes a husky bronchitic cough. The animals show a marked tendency to isolate themselves, and in cold weather seek warmth by burrowing under litter or huddling close to their companions; they frequently refuse to rise when recumbent, and at all times rise unwillingly and stiffly, and when made to move do so with a stiff gait, and soon show signs of exhaustion if submitted to exertion. The ears are frequently lopped, and not unfrequently present a congested appearance. The conjunctiva is injected and there is sometimes lachrymation or discharge of a small quantity of agglutinous mucus from the inner canthus of the eyelids.

"In the most advanced stages the above symptoms become aggravated and more pronounced, but their character will depend largely on the seat of the lesions. The temperature rises to 105-6-7 degrees F., the pulse becomes more rapid, the breathing hurried and, occasionally jerky, the urine scanty and highly colored at times, normal in character at others, but frequently evolving a peculiar characteristic odor. Wasting of the muscles proceeds apace, weakness increases, the movements become more unsteady, and often the hind legs are plaited one over the other. Food is refused, or only taken in small quantities, but there is frequently great thirst. The irregularity of the bowels is more marked, and the feces become of a dark color, pasty, and of a very unpleasant but characteristic odor. In the case of pregnant sows, abortion sometimes takes place as the disease advances, many of the young pigs being dead, some decomposed. Occasionally a little blood is discharged from the conjunctivæ or the nostrils.

"If the bowels are inflamed or ulcerated, diarrhœa replaces constipation, the discharges being of a dirty chocolate or of a yellow ochry

hue, and in any case they have an abominable odor, and are sometimes passed with a jerk, and, as the end approaches involuntarily. [Just the contrary is the case according to my observations. In fact, as I have shown elsewhere, the ulcerations in the intestines have no connection whatever with the choleroïd phenomena—B.] At other times the digesta is retained, accumulates in the large intestines, undergoes decomposition, liberates sulphureted hydrogen, producing tympany, which gives the animal a round appearance, and thus deceives the careless observer—it being taken for granted by such that the patient is eating well, and consequently that there cannot be much the matter.

“If the lungs are markedly affected, the breathing becomes labored as well as hurried, the cough (especially if animal excited) distressing; there may be catarrhal discharge from the nose and eyes, that from the latter causing dirt to adhere to the eyelids, thus giving the animal a very peculiar appearance. There is often a short, painful, hollow grunt, and on physical examination of the chest signs of consolidation are readily detected. The skin of the ears and abdomen is often cyanotic, and the extremities cold, and not infrequently foam (sometimes bloody) oozes from the nostrils, and when this takes place the breathing becomes gasping and oral.

“If the brain or cerebral meninges are involved the animal presents a stupid appearance, wanders aimlessly and mopeishly about, staggers like a drunken man, and often falls helplessly over, or if excited is thrown into a state of convulsions, on the passing off of which is left lying helplessly on its side. At other times his head is bored against a wall or thrust into a corner, or the nose is buried in the litter, producing stertor; the pupils of the eyes may be contracted or dilated, and the retina may be markedly injected.

“If the spinal cord or the meninges are affected there is marked muscular twitchings, chronic spasm, and partial, followed by total, paralysis.

“THE LOCAL EXTERNAL LESIONS

Are to be found in the skin. They consist of (*a.*) discolorations; (*b.*) vesication; (*c.*) pustulation; (*d.*) papulation; (*e.*) sloughs; (*f.*) desquamation.

“(*a.*) The discolorations vary from pale blue or light red to a dark, livid blue, purple, or black. At times a scarcely perceptible erythematous black is to be discerned on the skin of the abdomen, the vulva, the hocks, the ears, the throat, or the inside of the thighs; or it may be a little cyanosis (blueness) of the ears. In some cases this discoloration is diffuse, at others in discrete patches; when faint and undecided the animal should be excited a little, and the entrance of air to the lungs excluded for a short time; the discoloration of the

ears is rendered more distinct by taking the pig up by the hind legs. In many instances the undecided erythema during life becomes very pronounced and, chameleon-like, passes rapidly through various hues after the animal is stunned by a blow on the head.

"In the course of a few days—hours in some cases—the discolorations become more pronounced and more permanent; at first being due only to capillary congestion, they may pass off, but as venous congestion sets in, followed as it usually is by extravasation, the color becomes deepened, and the cutaneous structures positively stained, and if the pig recovers, it subsequently passes through the changes seen in ecchymoses from other causes, desquamation of the cuticle taking place as it passes off; on the contrary, the interference with the circulation is so great as to lead to necrosis, usually arterial and consequently dry and sloughing.

"In very many cases no pathognomonic discoloration exists; but even here it will be observed that the dermis over the lateral and inferior aspects of the body contracts a peculiar, yellowish-brown hue.

"Cyanosis is often the result of imperfect oxidation from the existence of extensive lung lesions.

"(b.) Vesication is rarely a primary lesion, and still more rarely is it a solitary lesion, being usually seen during the course of the disease, the vesicles forming in the patches of healthy skin, and being usually followed by desiccation and desquamation. The bullæ associated with sphacelus may be mistaken for vesicles.

"(c.) Pustulation, like vesication, is rarely primary or solitary, nor is it seen except in the most virulent cases. In some instances there is a distinct collection of pus under the epidermis, in other instances the apparent pustulation partakes more of the character of a circumscribed pemphigus, being marked by circular patches of a very dark red color, followed by the effusion of sanguineous serum and a little pus, the fluids drying and the superficial parts of the skin becoming necrosed, giving to the patches an appearance similar to that produced by the action of a powerful escharotic, such as nitrate of silver, or in some instances like the lesions seen in a virulent septicæmia.

"(d.) Papulation may be primary or secondary, it may be solitary or associated with other lesions. Very frequently, especially in old sows, the formation of papulæ is the first cutaneous lesion observed, and more, it is often the only skin lesion seen throughout the course of the disease. The papulæ vary in size from a hempseed to a pea; they are not usually of a very high color, and hard and unyielding, formed about the abdomen and thighs at first, and may appear in successive crops; as a rule, they fade away without going on to vesication or pustulation.

"(e.) Sloughs, as already indicated, are the result either of ab-

solute capillary or of hemorrhagic lesions. Sloughing is most largely seen in the ears, occasionally in the hamicles and the back; the tail is often lost by sphacelus, especially in very young pigs.

“(f.) Desquamation of the cuticle is not peculiar to the disease; it is a common result of all forms of fever. Icterus (jaundice) is not so frequently seen as it is in many other specific diseases. This may probably be due to the fact that jaundice is not often seen in the pig under any circumstances.

“DIAGNOSIS.

“The importance of a correct diagnosis in such diseases as the one under consideration cannot be too strongly insisted upon. If a false diagnosis is made, much injustice is done to the owner of the animals, much anxiety and often loss is caused, and the practitioner oftentimes brings down upon his head severe rebuke.

“The conditions most likely to be confounded with those characteristic of swine fever are—(a.) pneumonia with cyanosis; (b.) purpura hemorrhagica; (c.) urticaria; (d.) measles; (e.) variola; (f.) anthrax.

“(a.) Pneumonia is most likely to be mistaken for swine fever when it attacks a number of pigs simultaneously; when, as the result of bad hygienic surroundings or improper feeding, it is accompanied by diarrhœa, and when, from the involvement of a large lung surface, cyanosis is marked.

“(b.) In purpura hemorrhagica the discolorations of the skin, the congestions and extravasations, with the fatal character of the affection, are very likely to mislead. Purpura, however, is not accompanied by such a high degree of fever, is traceable to some local cause, as the ingestion of putrid or diseased flesh, bad drainage, or dirty sties, and is not contagious.

“(c.) Urticaria (Nettle Rash).—In urticaria there is a tolerably high degree of fever, derangement of the bowels, refusal to eat, isolation, tendency to seek warmth; there is frequently patchy erythema, and even discrete ecchymoses, with occasionally superficial pustulation, and sometimes a little hemorrhage from the conjunctiva and the nose, with conjunctival petechiæ, and in the sow vaginal petechiæ.

“The affection is sporadic and non-contagious; is traceable to indigestion of large quantities of cabbage, or to some other form of improper feeding; is not fatal and passes off in a period varying from forty-eight to sixty hours, though occasionally, owing to a renewal or prolongation of the existence of the determining cause, relapse occurs.

“(d.) The term measles in the pig is very frequently associated with hydatoid (*cysticerci cellulose*) disease. I do not refer to this. I am of opinion that the pig suffers from an eruptive affection in some respect similar to measles of the human nature. Measles often, how-

ever, attacks a number of young pigs; is accompanied by a moderately high degree of fever; by some is believed to be contagious, and if the animals affected by it are neglected or exposed to inclement weather, or damp or cold, they are very apt to suffer from broncho-pneumonic disease.

“(e.) Variola suillus is not often seen in this country, and, so far as I know, never occurs as an epizootic. It always runs a definite course, and the skin lesions are evolved in regular order. The characteristic intestinal lesions of swine fever are not developed, though there may be patches of congestion, or even of muco enteritis, discovered after death, with systemic effusion and even extravasation. As in swine fever, and in foot and mouth disease, the young frequently die while sucking their dams, without the latter giving any external evidence of the existence of such a disease in the system.

“(f.) Anthrax.—As before remarked, there seems to be an impression in the minds of some pathologists that because the pig resists experimentally the action of the anthrax virus he is not susceptible to anthrax. This is a great mistake and is not in accordance with clinical experience. Pigs frequently die from anthrax after the indigestion of the offal, the blood, or the flesh of cattle that have succumbed to the disease. The fatality of anthrax, the rapidity of its course, its contagious nature (unsuspectedly by indigestion), the marked blood lesions seen *post-mortem*, and the hemorrhagic condition of the lymphatic glands, all combine to lead the unwary practitioner astray. Anthrax is probably never seen on a farm as a primary affection in the pig, this animal almost invariably suffering secondarily to cattle; and the very fact that the disease has made its appearance in cattle first, or that the affected animals have been feed on offal or refuse from a slaughter house, is sufficient to enable an ordinarily intelligent practitioner to determine the nature of the malady without much difficulty. In any case where doubt exists, inoculation with a little of the blood of the dead animal — of some small animal — or microscopic examination of a drop of blood for the characteristic bacillus, will soon set the matter at rest.

“Finally, it may be said, that whenever a large number of pigs are found to be dying simultaneously (and especially extends over a large area), with symptoms of bowel or pulmonary derangement, and in some cases skin eruptions, it may safely be assumed that the disease from which they are suffering is swine fever. Practically, pigs in this country are not subject to any other fatal malady of an epidemic type.

“PATHOLOGICAL ANATOMY.

“In dealing with this part of the subject I shall direct attention (firstly) to the general conditions of the tissues of the body and the

blood; (secondly) to the pulmonary lesions; (thirdly) to the gastrointestinal lesions; (fourthly) to the lymphatic lesions.

"At the outset I may remark that many of the lesions of swine fever are not peculiar to the disease; they are only concomitants of it, and are common to the other affections in which the blood has undergone important physical and vital changes.

"The first thing to be thought of in making a *post-mortem* examination is to carefully look over the skin, not only in undressed, but in dressed carcass also, and it must be borne in mind that the cutaneous lesions will be materially modified by three circumstances:—(a) The fact as to whether the animal has died a natural death; (b) As to whether blood has or has not been abstracted at the time of or prior to death; and (c) As to whether the carcass has or has not been properly dressed, and the skin scalded and scraped. It must be patent to all ordinary observers that if an animal has been allowed to die a natural death with all the blood in its veins, or if it has been killed without the abstraction of blood, the skin lesions must be more pronounced than where opposite conditions exist. It must be equally plain that the removal of the bristles and epidermis, and with this the dirt from the skin, brings the skin lesions more prominently to view than is the case when the carcass is allowed to lie in the usual filthy surroundings of the porcine species. I do not, however, wish any person to accept these statements hypothetically. I assert that, after making hundreds of *post-mortem* examinations under every variety of conditions, and after having investigated the matter experimentally, the propositions laid down are substantially and essentially correct. Frequently, indeed, skin lesions are rendered prominent by dressing that could never have been detected during life.

"In the general condition of the carcass, much aid is not afforded to the inquirer. If the pig has been fairly well fed, if the disease has not been too long in existence, and if the carcass has been properly dressed and hung in a cool, dry atmosphere after dressing, even the experienced inspector can gain nothing by its physical examination. The fat may be as well set and as white, and the muscular tissues as dry and as firm, and of as good color and consistence as the carcass of a healthy pig, and even after hanging for several days these conditions may be preserved; if, however, the animal has been laboring for some time under the disease, if emaciation has set in, and if bowel or lung lesions are at all extensive, then the carcass will be pale, flabby, and moist, and suspicion will be aroused at once.

"The blood in the early stages, and even in the advanced stages, sometimes, does not present any important physical alterations. It may clot with tolerable rapidity and firmness and may be of its normal color; on the contrary, and in spite of the assertions of one or two of our best pathologists, it may present some very important

physical changes. It may be dark in color and thick in consistence, or it may have a tarry appearance, or, and more important than either, it may resemble a mechanical mixture of red brick dust, with some colloid solution. The last mentioned condition I look upon as very pathognomonic in its character, and I have frequently found such blood in the heart when I have failed to find sufficiently convincing evidence of the existence of the disease in any other organ or tissue of the body. *Ante-mortem* cardiac clots are sometimes found.

"The systemic lesions are extravasations of blood and effusion of serum and of lymph. The skin extravasations may be superficial or deep, they may extend into the subcutaneous tissue, or even into the subjacent muscles; they may be very dark or red in color, and usually become of a brighter hue after exposure to the atmosphere. They may be confounded with the lesions produced by bruising and the strokes of whips or sticks; the latter are superficial, and the extravasation is confined to the immediate seat of the injury. Along the back of the neck and on the back they may be confounded with the redness produced by the animal rubbing itself shortly before its death against a rail or spar; in this case the redness is diffuse; in swine fever it presents the defined characters of an eruption.

"In the muscular tissue itself the extravasations are rarely pronounced, but in the intermuscular connective tissue they are common, and very frequently, when doubts exist as to the nature of the malady, the detection of intramuscular extravasations, especially in the subscapular region, dispels the doubt.

"Serous or sub-serous extravasation is common into or under the peritoneum, into the pericardium, into or under the endocardium, particularly of the left ventricle, under or within the renal capsule, or the serous tunic of the liver or spleen, into or within the cerebral meninges. In form the petechiæ may be punctiform, ramiform, or diffuse; in color bright red or modena.

"The mesentery and omentum are more often the seat of congestion than of extravasation.

"Parenchymatous extravasation is sometimes very extensive, at other times absent; though visceral congestion, especially in the case of the liver, the kidneys, and the spleen, can generally be distinguished if carefully sought after.

"I have seen the kidneys presenting an appearance such as could scarcely be credited without ocular demonstration; the malpighian bodies as distinct as if they had been painted, the intratubular tissue mapped out to a nicety with effused blood, and the organ rendered more like a kidney into which blood had been injected with superhuman force, than anything else to which I can compare it. Usually the lesions are punctiform.

"Effusion of serum is found in the deep intermuscular connective

tissue, and into cavities, as the peritoneal, pleural, pericardial, and arachnoid; while layers of coagulated lymph are frequently found deposited in the serous surfaces of organs, causing adhesion of visceral to parietal reflections.

“Pulmonary lesions are by no means constant or necessary. They may, however, be primary, and may exist independently of intestinal or of any other lesions—frequently I have discovered (and confirmed by diagnosis thereby) pulmonary when the systemic and enteric lesions have been entirely absent. In the early stages the lesions, like those of pleuro-pneumonia, are of an effusive or exudative type. On careful examination patches of lung will be found in which the lobules have a more solid appearance and feeling than have the surrounding lung tissue, and in which the interlobular tissue is distinctly infiltrated with colorless serum, or more usually serum having a slightly sanguineous hue, and if a transverse section of the involved lung structure is made and pressure applied, serum oozes out in large quantities, and as it passes from the small bronchi it is seen to be mixed largely with air bubbles (frothy) showing that the effusion has been in existence during the life of the animal. In other cases small patches at the borders of the lungs are found presenting the characteristic appearance of small patches of consolidation from collapse.

“As time goes on the involved part of the lung assumes a condition very similar to that with which we are familiar in pleuro-pneumonia in the ox. The inflammation is circumscribed by effused lymph, the surface of the involved area is raised distinctly above the surrounding level, the pleura becomes opaque and thickened, and masses of lymph are found deposited on its external surface, not infrequently producing adhesion of the pulmonary to the costal pleura, while in very asthenic cases I have seen suppuration.

“On section the affected lung is found to be solid and friable, charged with serum, red, black, or grey in color (red, black, or grey hepatization) increased in density and in specific gravity, decreased in resilience, and, as in pleuro-pneumonia, separated from the surrounding lung structure by a distinct segregating band.

“The bronchial tubes, and often the trachea, are filled with serum or occluded with coagulated lymph.

“GASTRO-INTESTINAL LESIONS.

“In making a *post-mortem* examination of a pig supposed to have died of swine fever, the best plan to adopt is to carefully remove the whole of the intestines and stomach and lay them aside until the carcass itself has been examined.

“In most cases lesions in the small intestines are readily detected by visual examination; the coats being semi-transparent, any thickening or discoloration is readily seen from without as they run over the fingers.

"Adhesions of the bowels to each other, or to the mesentary or omentum, should also be noted, as such adhesions, when circumscribed, invariably mark the site of grave structural lesions in the walls of the bowels. Discoloration, ecchymoses, roughening and opacity of the peritoneum, are useful guides, as they are also in the localization of lesions. In the large bowels a peculiar blue discoloration, interspersed with petechiæ, usually capilliform, is often seen.

"The large bowels are best examined by making an incision immediately over the junction of the ileum with the cæcum and colon, and continuing the incision along these bowels, noticing first the character of the egesta they contain, and subsequently washing them, when any lesions which may be in existence are at once rendered prominent.

"Gastro-intestinal lesions, for convenience of description, may be arranged in two groups: 1. Those in which there are no marked necrotic changes. 2. Those in which such changes predominate.

"In the first group the prominent lesion is diffuse mucositis, extending frequently from the cardiac orifice of the stomach to the ileo-cæcal junction, and in some cases along the colon and cæcum.

"The inflammatory action may be very slight and extend no deeper than the superficial mucosa; on the contrary, it may be very pronounced, and involve the deeper structures of the membrane, which may become very thick, soft in consistence, and oftentimes of a gelatinous character. The epithelium in the worst cases is dry and roughened, and there may be superficial erosions. Extravasations into the structure of the bowels are often seen, and not unfrequently the contents are of a sanguineous hue. As a rule the fæces are either semi-solid or of a very liquid consistence, and give off a very unpleasant but characteristic odor, which lingers on the skin of the hands for a considerable time.

"In the second group very definite structural changes are observed; these are superficial necrosis in patches having an appearance similar to that which is produced by the application of cautery, and characteristic ulceration.

"Before describing the typical ulcers of swine fever, I wish to direct attention to an anatomical glandular peculiarity which exists around the base of the ileo-cæcal valve of the pig. If the surface of the mucous membrane is closely scanned a number of pin-like depressions or *cul-de-sacs* will be seen; these are probably the mouths of glands closely assimilated in character to the solitary glands. In the vast majority of cases the typical lesions of the disease are to be found associated with these pouches, *i.e.*, if there are any intestinal lesions in existence. The mouths of the sacs seem to be enlarged, and extending therefrom are masses of yellowish-brown-looking material, not unlike dried yellow putty; on the application of a little pressure these plugs may be enucleated intact, leaving behind a distinct alveolus with

jagged surfaces and edges, and often of a yellow, ochre hue. In the more advanced stages distinct molecular death has gone on, the ulcers have increased their boundaries by peripheral extension, and very frequently they have become confluent and form large, irregular patches around the base of the valve. Personally I attach much importance to these changes, and even where there seems to be an absence of bowel lesions I never fail to examine the part indicated.

"The typical ulcers of swine fever can scarcely be confounded with the lesions of any other malady. They are preceded by small nodular formations in the mucosa, these producing rounded elevations on its surface, and on passing the fingers over the part they can be distinctly felt; they resemble in size and general character the miliary nodes of glanders, and are probably the result either of plugging of the small capillaries (embolism) by bacteria, or of circumscribed inflammatory changes, leading to the throwing out of an exudate.

"In any case, they are of a greyish color, and at first are discrete and tolerably resistant to the touch. After a time central softening is observed to have taken place, followed by eruption and the formation of distinct, small, circular ulcers, with jagged edges, and of a greyish, greyish-yellow, or black color. Many of these ulcers remain discrete throughout the course of the subsequent changes; others run together, and many produce extensive, necrotic patches of a very irregular shape. Long ridges of necrotic tissue are sometimes arranged in parallel rows, giving the part the appearance of a crow's foot, as shown in preserved specimens in my possession. Whenever these extensive necrotic changes go on, the coats of the bowel, in the involved parts, become very thick and hard, and dark (either black or dark red) in color, and the patch stands prominently out from the surrounding bowel. In very severe cases there is a tendency to perforation, a result, however, very rarely seen, as it is prevented by adhesion of the investing peritoneum to that of an adjacent bowel, or to the mesentery or omentum. This condition is also well shown in specimens in my possession.

"The small ulcers already noticed tend to increase their bounds by peripheral extension, layer after layer of epithelium proliferating and degenerating, until ultimately ulcers as large as a six-penny piece or a shilling are formed, which, on close inspection, are seen to be made up of circumferential layers of necrosed tissue and cells, as seen in preserved specimens. In the course of time the necrosed tissues are cast off, but in the process of exfoliation the mass is sometimes seen to separate circumferentially after the manner of a varicose lesion, the central parts clinging to the subjacent structures with great tenacity.

"The necrosed structure is of various hues, but there is no doubt that the dark color so often seen is due to the dyeing effects of

coal and coal dust—a material ingested in large quantities, whenever opportunity offers, by the pig. The discoloration here and over the surface of the bowels is undoubtedly due in some cases, as in cattle plague, to the fact that the iron in the extravasated or stagnated blood has become converted into sulphide by the action of the hydrosulphuric acid so largely found in the intestines by the decomposition of the egesta.

“As already indicated, gastric ulceration is rare, and the ulcers do not partake of the specific characters seen in those of the intestines.

“The contents of the large bowels present remarkable characteristics. Sometimes they are simply fluid or semi-fluid, dark in color, and of a sickly but peculiar odor; but more largely they will be found (*a*) in the form of small, concrete masses, which adhere tenaciously to the mucous membrane, and when detached therefrom leave behind a roughened and sometimes an inflamed surface; (*b*) in the form of large masses of a very dark color, of firm consistence, very cohesive and coated with a layer of mucus or mucus mixed with blood.

“A very marked condition often associated with intestinal lesions is enormous engorgement of the mesenteric capillaries, and sometimes pronounced mesenteric extravasations.

“GLANDULAR LESIONS—LYMPHATIC AND MESENTERIC.

“There are perhaps no organs in the whole body which become so soon altered in their character as the lymphatic glands. Even when other evidence of the existence of swine fever is not forthcoming it can be obtained in these bodies. The mesenteric, bronchial, and hepatic groups are certainly the most frequently found to present the characteristic lesions, but even when all the viscera have been removed, there are still left for inspection the prepectoral, the sublumbar, the inguinal, and the laryngo-pharyngeal groups; and unless an animal has been slaughtered in the very earliest stages, they usually furnish us with indubitable evidence of the prior existence of the disease.

“The glands on section will be found enlarged and charged with serum, the cut surface being moist and glistening; the small vessels in the reticulæ of the lymph channels are found at the outset distinctly injected; but if the disease has been at all advanced, the injection gives place to extravasation, which, like the former condition, is confined at first to the lymph spaces; the parenchyma remaining intact and of its normal color (white) gives to the cut surface a mottled appearance—not at all unlike a section of the ‘Queen’ strawberry. After a time all structural characteristics are lost in the gland, it becomes universally infiltrated with blood, very large, very dark in color, and very friable. The conditions above described are character-

istic of swine fever; they are not, however, exclusively so; they are found in anthrax and in septicæmia, but, notwithstanding this fact, they afford us a very reliable piece of confirmatory evidence in all cases.

"The changes in the mesenteric glands are, on the whole, allied to those described in the lymphatics.

"TREATMENT.

"In view of the fact that treatment is not, except for experimental purposes, permitted by the privy council, it is unnecessary to make any extended observations in reference thereto.

"I may, however, remark that many years ago (over twenty) I used to treat cases of swine fever wherever and whenever they came under my observation; and notwithstanding the fact that therapeutics were not so far advanced then as now, I had a very gratifying amount of success in my treatment of the malady.

"The plan adopted was to clear out the alimentary canal by a purgative, following this up by the administration of sulphur and by potash salts; with ammon. carb. whenever there was a great depression or lung symptoms were urgent. Good, easily digested food, with plenty of milk was allowed.

"At the present day I should administer sodium sulphite and potassium chlorate; and if the fæces were very fœtid, sulpho-carb. of soda—alternating these agents with hydrargyrum cretæ, and preceding their use by the administration of a purgative.

"PREVENTION AND SUPPRESSION.

"I suppose the various legislative measures that have been put in force by the privy council are familiar to all those who are interested in the matter, and local authorities have large powers placed in their hands to enable them to cope with this porcine plague.

"Of the privy council measure, the 'Circle Order' is perhaps one of the best, and next in importance to this are the orders connected with the holding of fairs and markets. But, unfortunately, these measures are not, in most instances, put into force until the disease has made extensive ravages—until, in fact, the legend Iehabod may be appropriately written against them. This fault, however, does not always lie at the door of the powers that be. In reference to compensation and slaughter, it may be said that the measures now in force are only half measures. There is too much of the voluntary element about them to render them of any great practical value; and, as in the case of pleuro-pneumonia, there is no uniformity of action observed amongst different and adjoining districts.

"Whenever slaughtering is not decided upon, the first care of the inspector is to persuade the owner of the pigs to slaughter all animals

of whose carcasses anything can be made, and which are not of great value for breeding purposes. If proper precautions are observed, and perfect isolation and disinfection insisted upon, there is no necessity to slaughter the brood animals. The necessity for the immediate slaughter of affected, or even of suspicious, animals need not be insisted upon.

"In addition to these measures, the piggeries (every part of them) should be thoroughly cleansed and disinfected and painted over with fresh, hot limewash, mixed with a large percentage of phenol. I prefer myself gas tar to whitewash, especially when dealing with wooden erections. The floors of the sties should be put into thorough repair, and rendered impervious to moisture; the feeding troughs, if of wood, should be burnt; if of iron, cleansed, disinfected, and painted, and so arranged as to preclude the possibility of contamination by the feet of the pigs. Rats should be destroyed as speedily as possible, and if very numerous, liquefied gas tar should be forced into their holes and burrows, and a watch kept at the various outlets for the half stupefied rats as they make their escape therefrom.

"Aerial disinfection, by means of sulphurous acid gas and chlorine, should also be had recourse to; and medicinal disinfectants—hyposulphite of soda and potassium chlorate—administered to the healthy animals in their food.

"By the energetic adoption of the methods above recommended, the mortality may be considerably reduced, and the disease effectually suppressed; but while this is so, I do not hesitate to say that such measures are only temporizing measures, and that the most effectual method of suppression is the slaughter of all diseased animals and of those which have been exposed to the infection, with imperial and full compensation."

SWINE PLAGUE IN EUROPE.

The study of swine plague in Europe will be found a puzzling question to any one who undertakes to arrive at a clear and definite understanding by reading the literature upon the subject. While those who have studied the swine plague in the United States all agree in one thing, even to Mr. Salmon, that is, they all describe the same lesions, the only difference being that Detmers and myself can possibly find but one pathogenic germ, no matter how many slight variations may be seen in the necroscopical lesions, Mr. Salmon has attempted to make two diseases out of this one, and given a description of a microbe as the specific cause of the intestinal lesions in this disease, which it has been impossible for other investigators to discover. English investigators of undoubted ability also describe the same lesions in the swine plague of Great Britain as have been constantly seen in the disease of this country, thus completely putting any discussion of that question out of consideration. Cornil and Chantemesse have produced the same lesions in inoculated swine, and it is highly probable that the disease partially described by Rietsch and his colleagues is also the same, though we have not, at present, a sufficiently and detailed account of the lesions observed by them to positively assert that the diseases are all one of the same, but, as I shall show, the evidence is all in that direction. In a later paper, "Comptes Rendus," Tome CVI., Cornil and Chantemesse say, that they have since studied the symptomatology and etiology of the outbreak at Marseilles, and as they mention no points of difference we may conclude that they consider it the same disease they had already investigated at Gentilly. As has been already shown and will be still more effectually demonstrated, the investigators mentioned have all discovered apparently the same organism as the cause of these diseases, but, as I have also sought to make plain, too much stress cannot be placed upon this fact, for we also know that there is a disease known as rabbit septicæmia, as well as the hen cholera, and our southern cattle plague and the yellow fever of man, which are all caused by a micro-

organism that is a member of this ovoid, belted group. It might also be mentioned that the, so-called, sorghum disease of this country is also caused by one of the same species. Now, scarcely any sane man, with all these facts before him, should fall into the error to which Hueppe is inclined, of saying that all such diseases should be one and the same, because their micro-organismal cause looks to be the same; the clinical and necroscopical phenomena too positively contradict any such opinion to necessitate discussing it at present. However, there is no question that nearly all the investigators of these porcine pests have been inclined to fall into this error, and nothing has prevented my making the same mistake but the fact that the clinical and necroscopical phenomena too strongly opposed such a procedure and suggested caution; still I have been near enough falling into the same error, notwithstanding a constantly increasing skepticism in my own mind. This tendency has all been in one direction, viz., the endeavor to find support in the investigations of Loeffler and Schütz, of Berlin. Had it not been that Schütz himself has been more than half inclined to accept Roloff's description of lesions observed by him in swine, as far back as 1875, as belonging to the Loeffler-Schütz "Schweinesuche," I should not have been at all inclined to accept the investigations of these observers as applicable to the American swine plague. This leaning upon Schütz especially, who defined the disease observed by him as an "infectious pneumonia," has been the stronghold of Mr. Salmon in his attempt to differentiate the swine plague of this country into distinct and separate intestinal and a pulmonary complication. These things taken together, and the fact that a swine plague has been described as newly appearing in Sweden and Denmark, for which there are strong grounds of assuming it to be the same as that of this country and Great Britain, has led me to a most exact and critical review of the foreign literature, at my command, upon the subject. I will here remark that I am disinclined to the opinion that the differences between the micro-organism described as causing the disease in Sweden and Denmark, and that of Loeffler-Schütz, are as striking as Selander has said, for if this be so, then the disease in those countries is certainly not that of Great Britain and the United States, the micro-etiological organism of which presents no marked differential characteristics from that of Loeffler and Schütz, as described by them and illustrated by the latter. In mentioning the diseases of animal life

that are known to be caused by different members of this ovoid, belted group, I have purposely omitted one to which I now desire to call especial attention, for its importance in settling this identity question cannot be overestimated. I allude to the "Wild-seuche" of Germany; a disease which manifestly differentiates itself from the cosmopolitan swine plague, both by its clinical phenomena and the absence of the, so-called, characteristic ulcerations and neoplasms of swine plague in the intestines. It derives its name from the fact that attention was first called to it by its decimations among the deer of the royal reserves in various parts of Germany. It distinguishes itself from swine plague by also attacking cattle, which the former has never been known to do. Like the swine plague, it is characterized by pulmonary lesions, but, as said, any specific intestinal lesions seem to be wanting, and it is scarcely probable that lesions so characteristic would not be seen in the longest known and probably best studied of the swine diseases of Germany. Whether or not this "Wild-seuche" also occurs all over Europe is a question that cannot be decided at present, as also whether or not it occurs in this country, but as has been said, there are diseases, here in the West, which we know nothing about, and one of which has been but briefly alluded to, that is caused by a germ of this same ovoid, belted group.

I wish now to call the attention of my readers to this "Wild-seuche," and to offer to their consideration a translation of the best description of it at my command; as said, its importance to the question in point demands that we know all we possibly can about it.

"THE WILD UND RINDER-SEUCHE.*

"Under the above name, Bollinger—1878—described a pest which appeared among the deer of the Royal Parks in the vicinity of Munich; it also occurred as an enzootic among cattle at the same time, as well as extended to horses and swine. Three hundred and eighty-seven deer and 234 wild swine perished at one outbreak. It again appeared in the next year, and was especially devastating in the summer of 1881. It has broken out in Prussia and other parts of Germany. It is not a new disease by any means, but has been mistakenly complicated with anthrax.

"*Ætiology*: In the year 1885, Prof. Kitt, of the Munich Veterinary School, demonstrated the presence of short, thick organisms in

*Lehrbuch der speciellen Pathologie und Therapie der Hausthiere, by Friedberger and Froehner, 1887, Vol. II., p. 423.

the blood and other organs of cattle that had perished; these micro-organisms colored only at their pole ends. Their pathogenetic action was proven by inoculations upon mice, rabbits, pigeons, birds, swine, goats, horses, and cattle. The bacteria resembled those of rabbit septicæmia. He found similar organisms in old specimens that Bollinger collected in 1878.

"Hueppe considers these bacteria to be identical with the Loeffler-Schütz organism, though this is no proof that the diseases are one and the same.

"Pathological Anatomy: From this point of view the disease may be said to appear in three forms: an exanthematic, a pectoral, and an intestinal; the two former are the most important and often complicated with the third; in some cases all three are present.

"1. The exanthematic form manifests itself by an enormous tumefaction of the skin and the subcutaneous connective tissue. The skin of the complicated parts (head, intermaxillary spaces, neck, etc.) is œdematously swollen, and the subcutaneous tissue enormously distended and filled with a sero-gelatinous material, which fills the meshes of this tissue so that, on section, a clear or amber-colored fluid oozes out; hemorrhages in the same are frequently present. The neighboring lymph-glands are also in the same condition and excessively swollen. The mucosa and adjacent tissues of the oral cavity also show like changes. The tongue is often so swollen as to be monstrous, its surface being of bluish-red color or dirty, brown color, the surface eroded and its tissues infiltrated with a sero-hemorrhagic or clear fluid. Similar changes occur in the pharyngeal mucosa. The salivary glands are dry and anæmic. The mucosa of the respiratory tract can also be swollen in the same way. Hemorrhages are present in the serosæ, in the muscles, lungs, etc. The spleen is perfectly normal [!?—B.] and the blood generally of normal color and consistency. Hemorrhagic enteritis is generally present also.

"2. In the pectoral form the interstitial connective tissue is infiltrated with serum, and the lungs hepatized. The pleura is inflamed and swollen, and covered with sero-fibrinous or fibrinous exudations. Effusions in the thoracic cavity are frequent; pericarditis and mediastinitis frequent; numerous hemorrhages and hemorrhagic enteritis also accompany this form.

"In the intestinal form the mucosa, especially that of the small intestine, is swollen, the epithelium desquamated, and the mucosa the seat of more or less extensive hemorrhages; the contents of the intestines are thin and bloody. These changes seldom occur alone, but generally complicate the pectoral or exanthematic forms."

It is much to be regretted that this description is taken from cattle only, as it would be very advantageous to have the same with regard to the disease in swine, in order to compare them with the necroscop-

ical notes of other observers in what has been termed "*Schweineseuche*." What I desire to call especial attention to, however, is the marked and enormous œdema which is described as complicating nearly all parts, as well as the constancy of the hemorrhagic lesions. That the disease is not the malignant œdema is shown conclusively by the facts that the "*Wild-seuche*" is conveyable to healthy animals, both by feeding and inoculation, while the former can only be imparted by inoculation.

It is not necessary to my purpose to notice the clinical symptoms of this disease, for they can be fully appreciated by considering the above described lesions. One point may, however, be mentioned, viz.:

"The animals suffer from tenesmus, groan, and lie down frequently, and even pass off croupous casts with the fæces, which later on become diarrhœaic. In cattle the course of the disease is from twelve to thirty-six hours for the exanthematous, while the pectoral form is longer, sometimes extending to five or six or even eight days."

The questions which now demand our attention are :

1st. Is the Loeffler-Schütz "*Schweineseuche*" the "*Wild-seuche*" or not?

2d. Is this "*Schweineseuche*" identical with the American and English swine plague and that described by Cornil-Chantemesse and others in France, and Selander and Bang in Sweden and Denmark?

To my mind we shall find that the evidence given by Loeffler is most decidedly affirmative with reference to the "*Wild-seuche*," and equally negative as regards the cosmopolitan swine plague, while that of Schütz partially tends in one direction and partially in the other. The morpho-microscopical, or even biological resemblances of the etiological organisms are utterly valueless in deciding this question, as has been intimated, and will be more completely demonstrated in another place.

LOEFFLER'S EVIDENCE.

While engaged upon investigations of the "*Rothlauf*," 1882-3, Loeffler says :*

"On the 26th of October, 1882, Dr. Eggeling, of the Berlin Veterinary School, placed at my disposal a hog which had died at the

* Arbeiten a. d. Kaiserlichen Gesundheits Amtes, 1885.

swine market, and which he said had perished of 'Rothlauf,' or erysipelas.

"Autopsy: The skin of the abdominal region and in the vicinity of the sexual organs was of a livid red color. Enormous œdema of the skin of the neck extended between the fore legs to the forward part of the abdomen. Pharynx reddened and swollen. The mucosa of the larynx and trachea of an intense, dark red color. But slight changes in the lungs; some portions of the right lung were dark red and somewhat atelectatic. Nothing especial to be seen in the heart. Liver and kidneys clouded. Mucosa of stomach intensely red, as well as that of the anterior portion of the duodenum. The balance of the intestine unchanged. Mesenterial lymph-glands unchanged. Spleen swollen and of a dark blue color; quite dense. The organs were still warm. Pieces of the same were at once placed in glasses that had been previously cleansed and disinfected, the pieces of the skin and intestines by themselves.

"Cultivating media were at once sown from the œdematous skin, liver, and kidneys. On the following day a development appeared in each of the glasses. When examined microscopically it was found that but one variety of bacteria had developed in each culture. These were exceedingly small, ovoid bacteria, which reminded one of the germ of rabbit septicæmia (see Plate III., Fig. 6), especially those observed in the process of fission, yet they were to be distinguished from the latter by not being over half as large.

"Various animals were inoculated from these cultures; the mice presented œdematous swelling of the sub-cutis. * * * In all organs the same bacteria. In guinea-pigs the locus inoculationis felt œdematous; the results of the autopsy was the same in all cases; hemorrhagic-serous infiltration of the sub-abdominal sub-cutis; the muscles of this region were also filled with a reddish, œdematous fluid. Lungs grey-red. Spleen scarcely swollen. The same bacteria, and only these, were found in the microscopic examination of the liver, the skin, the kidneys of the swine as in these animals." *Arbeiten a. d. Kaiserlichen Gesundheits Amt., 1886, pp. 51-2-3.*

From these animals cultures were made, and from these still other animals were inoculated, with the same results. The one characteristic phenomenon was, "Enormen, blutigserösen Ödemen der sub-cutis und blutigseröser Durchtrankung der Muskulatur des Bauches," or "(Edem von der Impfstelle ausgehend," that is, enormous œdema of the sub-cutis and a sero-hemorrhagic infiltration of the abdomen, or œdema extending from the place of inoculation.

These results of Loeffler's instigated me to especially repeat the same upon rabbits with pure cultures of the germ of swine plague.

The following notes will demonstrate the difference in the diseases at once.

From fluid culture of a pig that was killed in the laboratory on account of swine plague, I inoculated a rabbit and ground squirrel at ten A.M. on the 14th of May, 1888, with no very manifest signs of illness until the evening of the 16th; the rabbit died about 12 on the 17th, and the squirrel about 3 P.M. the same day. While I have tested many cultures on these animals I had not considered it of importance, previous to these critical studies, to record the autopsies, for to my mind, they had nothing sufficiently characteristic about them but the phenomena to be expected in an acute blood poison disease.

As the necroscopical phenomena were the same in both rabbit and squirrel, I will only record those of the former. As said, my interest in the patho-anatomical results in rabbits with pure inoculations of the germ of swine plague was re-awakened by the result of my very exact and critical studies of the investigations of Loeffler and Schütz. By reading the testimony of these observers it will be seen that "enormous œdema" and more or less extensive hemorrhages about the body of these inoculated animals were the essential characteristics. My two years very varied experiences with the American swine plague have shown me the total failure of the former, as well as that the latter depends on the acuteness and virulence of the attack. In this case I did not expect it, as the pig from which this material came had a very mild attack, and as it was only three weeks old the chances are that it may have become infected through the milk of the mother, which was sick of swine plague at the time; the rabbit thus may have represented the third generation of the germ in passing through animal organisms, and I know that this microbe becomes weakened in that way, even in passing through, or being passed through, swine, which mitigation again depends upon the virulence of the original microbes when the primary animals are infected from their pens, etc.

The rabbit in question was inoculated with one-half ^{ccm} of a bouillon culture of the swine plague bacterium, in the sub-cutis of the inside of the right thigh. During the first two days there was some swelling, heat, and diffuse redness at the locus inoculationis, but this had entirely disappeared at the time of making the autopsy.

Autopsy: Male rabbit, six months old, white in color, no swelling

at point of inoculation ; veins of sub-cutis engorged, superficial lymph-glands swollen, juicy and full of hemorrhagic points, resembling those spoken of in the autopsies upon swine. Vessels more injected in the vicinity of the point of inoculation, where the skin was of a pale yellow color, but nothing resembling œdema was present. (I wish it to be distinctly understood that I do not deny that some œdema may be present, but only circumscribed, when a more virulent culture is used. Never enormous œdema, however.) On opening the abdominal cavity, the vessels of the mesentary were repletely engorged, as well as those of the serosa of the small intestine, and to a less degree those of the large ; mesenteric lymph-glands swollen and hemorrhagic. Peritoneum normal.

Stomach : full of finely masticated food ; mucosa swollen and covered with a viscid coating ; towards pylorus stained yellow. In the vicinity of the œsophageal opening a small, irregularly-shaped, hemorrhagic center, covered with a yellowish, dry, caseous material, which adhered quite firmly, but on removal showed an ulcerated, granulous surface beneath. Mucosa of small intestine swollen and catarrhish, contents semi-fluid ; duodenal stained yellow ; contents of large intestine pultaceous, mucosa normal, but the vessels of the transverse folds were deeply injected, no indications of ulceration present.

Spleen : swollen and full of blood ; pulp soft.

Liver : swollen, full of blood, parenchyma of greyish-red color, which in many places was interrupted by a shade of yellow ; acini distended, some red, some grey-red, some yellowish-red.

Kidneys : swollen, capsule non-adherent ; cortical substance swollen, semi-opaque, greyish-red in color ; medullary dark red, vasi-recti engorged and extending in same condition into cortex.

Pelvic cavity : lungs comparatively normal, outer surface somewhat darker red in color in small spaces than the balance. Bronchial lymph-glands as others. Heart : ventricles contained coagulated blood of a dark red color. Myocardium opaque, greyish-red in color, and swollen. Ovoid, belted germs in all organs and blood, as well as found in the contents of the large intestine.

From the previous rabbit, which died on the seventeenth of May, 1888, I inoculated another, two-thirds grown, white, male rabbit, with an emulsion of the spleen of the former rubbed up in freshly sterilized water. One-quarter ^{ccm} of this mixture was injected under the skin

of inside of the thigh of the right leg, and five drops under the skin of the right ear. While the latter became of a diffuse redness, with the larger vessels engorged, there was no swelling of the organ, nor of the surrounding tissues. The only indications of illness were an increasing want of appetite to the twenty-third, when the animal was found dead in its cage at 9 o'clock. It was inoculated at 3 P.M., on the 17th.

Autopsy: Blood, dark red in color.

On removing the skin the subcutaneous vessels were seen to be engorged, at and around the locus inoculationis they were more so, and the skin was of a diffuse, yellowish-red color, but no œdema of the sub-cutis or adjacent muscles and no extravasation of any kind. The lymph-glands of the neck, the axillary and the external inguinal region were swollen and contained some hemorrhagic spots. No effusion in abdominal cavity. Peritoneum of abdominal wall normal; that covering intestines normal, except that of the large, where the vessels were engorged, especially those of the mesenterium; lymph-glands as above.

Stomach: no diffuse redness, or hemorrhage; distended with food, mucosa swollen, and covered with a viscid coating; cardiac portion of a diffuse redness. Intestinal mucosa somewhat swollen and covered with a viscid coating in the small, while in the large it was normal, some engorgement of the vessels of the transverse folds of the colon. Spleen swollen, dark in color, and soft. Liver swollen, the lower ends of the lobes very full of blood, while toward the base there were many anæmic patches, which were opaque; acini much distended, and of a yellowish-red color. Kidneys very much swollen; capsule non-adherent, cortical substance opaque, yellowish-grey-red in color, many vessels engorged at joints, some glomeruli visible as small red points; medullary substance marked by a dark, blue-red line on its cortical limits, which became paler as the pelvic mucosa was reached.

Thoracic cavity: no effusion; bronchial lymph-glands as above; right lung full of dark red spots of collapsed tissue, with many spots of a less red color, and still more dense. On cutting open this lung, a reddish, aqueous fluid oozed from the surface; left lung normal. Heart: pericardial sack contained a small quantity of a yellowish fluid. Myocardium opaque, yellowish-grey-red in color, and soft in consistency. As the bladder was full I thought I would test the

urine, and found considerable albumen in it; granulous tube casts were found quite plentifully represented on microscopic examination. The "belted, ovoid" germs were also found in small numbers in the urine, and in more replete representation in the blood and from the organs in covering-glass specimens.

Although the previously quoted autopsies present that constancy of patho-anatomical phenomena which should convince any one that the rabbits were not affected with the "Wild-seuche," and, hence, that that disease is not identical with the swine plague, the "enormous œdema" being entirely wanting; still, as an accident placed in my hands some excessively virulent material from swine plague, it seemed best to re-test the question by another inoculation of a rabbit. On Wednesday, May 23d, at about eleven o'clock in the morning, Regent Dr. George Roberts came into the laboratory with cultures and some blood, two days old, which he got from an excessively virulent outbreak at Creighton. Of the hog from which the material came, Dr. Roberts said the blood was thick as tar, and the superficial inguinal glands so swollen that their location could easily be seen through the skin. All the lymph-glands were of a dense, diffuse, blue-red color, the spleen swollen, the organs excessively hyperæmic. No consolidation in the lungs, or ulceration in the intestines. The ovoid, belted germ was present in a profuse representation in the blood and organs, and was also in the cultures brought to me by Dr. Roberts. In the bottle which he brought was a dark, purple-red fluid, in which was some thicker semi-coagulated material of the same color. On opening the bottle a slight odor of putrefaction made itself apparent, but the microscopic examination revealed innumerable bacteria of the ovoid, belted group, some streptococci, and some fine, short bacilli. At four P.M. of the 23d, I inoculated a six-months-old, white, male rabbit with two drops of this material, in the right ear, near the base, and five drops on the inside of the left thigh. On the morning of the 24th the lower portion of the ear was of a diffuse redness, with engorgement of the larger vessels; same, but to a less degree, of the inside of the left thigh. No œdema in either locality. Animal still eating, but not very ravenously. No change on the evening of the same day, no change in deportment. Nine A.M. of the 25th, rabbit dead. Rigor mortis pronounced. No change in the appearance of the loci of inoculation, so far as outside appearances went. On re-

removal of the skin the subcutaneous vessels were much more deeply engorged than in either of the other cases, and looked like blue-red lines or cords, according to size. The blood itself was of the same color and but partially coagulated. On opening a superficial vein, under due precautions, and preparing covering-glass specimens, this blood was found to be literally swarming with the ovoid, belted germs, and no other. At the locus inoculationis of the left thigh one might expect to find some œdema under such circumstances, but the point was marked by the absolute absence of the same, both in the sub-cutis and adjacent muscles. On the contrary, along the line of the large vessels and nerves, there was a slight collection of pus of a reddish-yellow color; small hemorrhagic centers were present, both in the muscular aponeurosis and in the tissues of the superficial muscles, but not in those more profoundly situated. The conditions in the vicinity of the base of the right ear were but an exaggeration of those already described. No œdema present!! The lymph-glands in the vicinity were swollen and of a diffuse redness; cut surface moist and glistening. The abdominal cavity did not contain any effusion, but the peritoneum was much swollen and very glistening, and covered with the most delicate, colorless coating of a viscid character. Vessels of the serous covering of the intestines intensely engorged, but neither diffuse redness nor hemorrhagic centers were to be seen, but on the great curvature of the stomach these conditions were more excessive. Mesenteric vessels engorged; lymph-glands swollen, and blue-red in color. Stomach contained a considerable quantity of a dark green ingesta, grass. Cardiac portion swollen, vessels injected, many localities of a diffuse redness, interrupted with numerous hemorrhagic centers; a viscid material covered the whole. Inside of intestines same as in previous autopsies. Spleen very much swollen; pulp purple-red in color and replete in blood; ovoid, belted germs present in profuse quantities. Liver much swollen, outer surface smooth, but dotted with innumerable centers of grayish-yellow-red color; gall bladder distended; inside surface moist and glistening, of the same variegated appearance, but in general opaque and greyish-yellow; interacinous vessels engorged, acini swollen, some of a dark red color, others yellowish and opaque. Kidneys as before, except more diffusely red in all parts; still cortex, marked by its opaque, greyish color, even though more injected. Lungs quite full of blood, mottled by red centers, cut sur-

face dry. Bronchial glands same as before, as well as those along line of posterior aorta. Tracheal mucosa swollen, and one diffuse, purple-red in color, in which could be seen the larger vessels still darker than the general tinction.

Heart as before.

This evidence should be sufficient to prove two points:

1st. That swine plague, like the Wild-seuche, is a "septicæmia."

2d. That it differs from the Wild-seuche in that its micro-etiological organisms do not cause "enormous œdema," nor is the tendency to excessive hemorrhage so great.

Of the Wild-seuche, Loeffler says:

"Notwithstanding 'die Haut am Bauche an den Geschlechtsteilen und am Hals rötlich livid,' (the skin of the belly and in the region of the sexual organs was livid red,) still this disease was not the 'Rothlauf,' or erysipelas."

Also:

"From these observations it is to be seen that the bacteria found by me in the fresh organs of swine that had died from a disease bearing very marked outward resemblances to 'Rothlauf' (erysipelas), are entirely different in form, development, and in cultures, as well as in their pathogenetic action upon different animals, from the small rods found in typical cases of Rothlauf." (See Plate III., Figs. 3 and 5.) *Ibid.*, p. 51.

If we now compare these results of Loeffler with the description given by Friedberger and Froehner of the necroscopical lesions met with in the "Wild-seuche," we shall find the most exact agreement except in one particular, and that is, that these authors tell us that the spleen is not swollen, while Loeffler mentions such a condition as present. The same variation will be found in different cases of the cosmopolitan swine plague, the spleen being sometimes intensely engorged and swollen, and at others not so, which is dependent entirely upon the acuteness of action of the toxin, and its effects upon the circulation. As has been said, F. and F.'s description has reference to cattle only, and as we are in want of necroscopical observations from other authors, especially on swine, and as we know that a similar, but not the same, organism does cause swelling of the spleen, and at other times does not, in our swine plague, it should be evident that too much value cannot be placed upon one single phenomenon. It will be observed,

on the other hand, that no mention is made of anything pertaining to the "characteristic lesions" of swine plague proper, as so frequently seen in the intestines.

Now, why not the swine plague, as we understand the term?

1. Because enormous œdema and deformation of the body thereby never occurs in that disease.

2. Because the tendency to hemorrhagic effusions is not a constant phenomenon in swine plague, though frequently present, but seldom to such an extended degree as in this disease.

3. Because the swine plague pneumonia is of a bronchial or destructive caseous type, which is not mentioned in this disease by these observers.

4. Because the "characteristic intestinal lesions" seem to be entirely wanting.

Hence it seems as if our conclusion is correct, that the organism discovered by Loeffler in swine, and the disease resulting from its action, has no authoritative bearing whatever upon the origin and nature of the cosmopolitan swine plague, though of essential value in showing that both diseases are septicæmiæ of an extra-organismal origin. This conclusion, that the disease described by Loeffler must have been the "Wild-seuche," is ably supported by Hueppe in his consideration of this question.

Hueppe describes the exanthematous form of the "Wild-seuche" as follows :

"The exanthematous form of the "Wild-seuche" bears a strong resemblance to the carbuncular, erysipelatous form of anthrax, and has been observed in cattle and swine. Within four to six hours there develops an acute inflammatory disease of the skin, which becomes swollen so intensely as to be deforming, and is also as hard as a board; this œdema also extends to the soft and pendulous portions of the body, which become infiltrated with a serous or serous-hemorrhagic effusion. When the head and neck of the animal are complicated we may see the most colossal swellings, which deform the parts to an excessive degree; the mucosæ of the head are cyanotic and filled with a hemorrhagic effusion. This form is undoubtedly due to traumatic infection, the virus gaining access to the blood, thus giving rise to a genuine septicæmia." Hueppe, Berliner, Klin. Wochenschrift, 1886, p. 754.

"Professor Kitt, of Munich, was the first to demonstrate the micro-etiological organism of the 'Wild-seuche.' He considered 'these

germs to be similar to, but not identical with, those of rabbit septicæmia, hen cholera, and swine plague.'” Ibid., p. 763, 1886.

Hueppe himself says:

“The blood contains the bacteria, which are demonstrable both by the microscope and cultivation many hours before the death of the animal. When the blood is examined the greater number of the bacteria appear as short rods, which are two to three times as long as wide, and have strongly rounded ends, the poles coloring very sharply, while the middle piece remains clear. * * * The formation of endogenous spores certainly does not take place. * * * When cultivated in gelatine they appear as isolated white colonies, while on agar they have a gray-white color. * * * Upon potatoes they form a greyish-yellow coating.” Ibid., pp. 756, 757.

It will be seen that this contradicts Loeffler’s statement as to these germs not growing on potatoes. Hueppe is correct, however, and there is scarcely a doubt that every variety of these germs will grow upon potatoes.

PROFESSOR SCHÜTZ’S EVIDENCE.

A careful consideration of the investigations of Professor Schütz will be found to complicate the question under consideration a great deal, for, while he undoubtedly gives evidence which shows that some of the diseased swine examined by him must have perished from the “Wild-seuche,” he gives equally good testimony that some of the others displayed necroscopical changes which look much more strongly towards the cosmopolitan swine plague. It was these latter cases, and the fact that the notes of the pulmonary lesions as given by Professor Schütz, when taken together with those described in the intestines by Roloff, completed the patho-anatomical picture of the true swine plague, that previously inclined me to the error, though in a less degree, of Hueppe on the one side, and Salmon on the other, that the “Schweineseuche” and the swine plague of this country must be identical diseases. The similarity in the bacteria largely supported this hypothesis, but this idea was essentially demolished by my studies upon the southern cattle plague, and the results of my examinations of the yellow fever material at my command. Even before these latter investigations had been thought of, I had taken a positive stand against Hueppe’s attempts at generalization, based upon the resemblances in micro-organisms alone. If my conclusion, or perhaps

better, hypothesis, becomes confirmed, it will be seen that Schütz fell into the same error as the rest of us, and, because the germs discovered by him in all the animals examined were apparently the same, did not give sufficient attention to the variation in the necroscopical lesions. His want of any practical acquaintance with the outbreaks from which his swine came, or even with the swine diseases of Germany, is self-evident from his own writings, and this failing of a very fundamental necessity to an accurate knowledge of these diseases on the part of the Germans is admitted by Froehner and Friedberger.—(l. c.)

Schütz opens up his observations as follows:

“Dr. Loeffler has used the names ‘Rothlauf’ (erysipelas) and ‘Schweineseuche’ (swine plague) in the sense that the disease caused by the fine bacillus is the Rothlauf (Plate III., Fig. 5), while the disease in which this other bacterium is found is the Schweineseuche. I shall use the differentiation, or nomenclature, in the same sense in the following remarks.” *Ueber die Schweineseuche. Arbeiten a. d. Reichsgesundheitsamte, 1886, p. 376.*

Of the micro-organism discovered by him as the cause of swine plague, Schütz says:

“The bacteria have an ovoid form, and are very easily colored in methylen-blue, gentian-violet, etc. When colored in a solution of gentian-violet they show an uncolored space in their center which is surrounded by a layer of colored substance. The quantity of this colored substance is greater at the poles of the organism, so that its ends appear more strongly colored than the middle part. When strongly colored they appear of a homogeneous blue.

“As these objects occupy an intermediate position between micrococci and bacilli, they should be looked upon as bacteria.

“They are 0.0012 mms. and 0.0004 to 0.0005 wide; their length is from one-third to one-half that of the diameter of red blood cells of the mouse.

“They proliferate as follows:

“At first they increase in length and become about double as long as wide, and have distinctly rounded ends, and color in the same manner as the micro-organism in septicæmia in rabbits, so that from a third to a half of the body presents itself as an uncolored space between the distinctly colored ends. More exact observation shows that the colored ends are connected together by a band of the same substance extending along the sides of the organism.

“The ends become separated from one another by the disappearance of the middle piece. They at first appear round, but soon assume an oval form, consequently two individuals are developed from each organism by fission.

"When this process proceeds rapidly, as we have found to be the case within the porcine organism, the microphytes do not attain the above given dimensions, but divide themselves so rapidly that the uncolored middle-piece cannot always be distinguished. Under these conditions the successive generations become smaller, which is to be attributed to slower development. The younger generations are often exceedingly small, but are still distinctly oval, and color profusely." p. 380-81. (See Plate III., Figs. 3 and 4.)

"When inoculated upon meat infusion, peptone-gelatine, one observes the development of numerous white points which either lie in groups or isolated colonies. The points gradually augment in size until they coalesce with those in immediate proximity, so that the inoculation puncture appears as a homogeneous line with punctiform excrescences along the sides. A grayish-white wall develops around the point of inoculation upon the surface of the gelatine.

"The organism does not cause the gelatine to become fluid." p. 382, l. c.

It can be readily seen that this micro-organism of Schütz exactly corresponds to the one described by me in its deportment in gelatine, and exactly in its microscopical and morphological appearances, when stained.

We will first show that Schütz accepts Loeffler's conclusions as to the necroscopical lesions of their "*Schweineseuche*," which, as said, we believe to be the well-known "*Wild-seuche*," and then show that some of the animals examined by Schütz presented the same phenomena. He says, quoting Loeffler:

"The skin of the abdomen, the sexual organs, and neck was livid red in color. Enormous œdema of the skin of the neck, which extended between the anterior extremities and along the abdomen; pharynx reddened and swollen. Lungs but slightly affected, on the right side some dark red spots; mucosa of trachea and larynx of an intense, dark red color. Heart, nothing special. Parenchyma of liver and kidneys swollen. Mucosa of stomach and anterior portion of the duodenum intensely red; balance of the intestines unchanged. Mesenteric glands not enlarged. Spleen somewhat swollen, dark red, and very consistent." *Ibid.*, p. 376.

That the above description does not apply to the swine plague must be self-evident to every one properly acquainted with that disease.

Of his experiments which point towards the "*Wild-seuche*" as one of the diseases investigated, Schütz says:

"On June 15, 1885, I received the stomach, spleen, and liver of three swine of which it was reported that they had perished from the Rothlauf."

It will be observed that the German veterinarians were totally unable, at this time, to distinguish between the two diseases from the clinical stand-point. As the organs had undergone some cadaveric changes, a report of their condition is not given, but Schütz says:

"That the examination of covering glass specimens did not reveal the presence of the fine bacilli of the Rothlauf (Plate III., Fig. 5); on the contrary, numerous other organisms were seen, among which was one variety having an oval form, which at once called to mind those described by Loeffler as occurring in the Schweineseuche."

A pure cultivation of these was secured by the inoculation of some small animals; of the necroscopical results we will note but a few remarks. In this case the "œdema" was the characteristic feature.

"Upon mice the obduction showed the subcutaneous tissue to be œdematous and bright red. Lymph-glands of the knee-fold [Salmon borrows this term in his latest report] swollen, moist, and dark red. Large intestine well filled with fecal mass.

"Rabbits, inoculated on right ear, which became swollen, painful, hot, and red, this tumefaction extending over the entire head and neck; the sub-cutis of these parts was filled with a clouded fluid; this œdematous condition extended through the respiratory tract to the lungs. Spleen swollen, soft, and brown-red."

Other autopsies upon these animals gave similar results. We will now turn to the swine inoculated with pure cultures obtained from these animals. I will not give the history of these experiments, but will at once proceed to quoting portions of the necroscopical notes:

"Two pigs, five months old, inoculated June 26, at about 5 P.M., died between 6 and 7 P.M. on the 27th, 26 hours. [This looks like the Wild-seuche, and not at all like the swine plague, as the inoculation was subcutaneous. As can be seen by referring to my autopsy notes, in only one case did I receive any such rapid result. In our swine plague, even by intra-abdominal injection of cultures of the first generation, or even material directly prepared from the spleen in distilled water, death seldom results in less than 7 to 10 days, and frequently extends to 20, which was the case in the Cornil-Chantemesse experiment, where the bouillon was injected directly into the lungs. It may be well to mention here, that in this case the "characteristic tumors" were found in the intestines, which will be most effectually missed

in all Schütz's cases, though we do not consider them an absolutely essential characteristic of the swine plague, but they could scarcely be wanting in so many cases as Schütz examined had the animals all died of the same disease.] The changes in the first pig were, the inner surface of the thighs and posterior part of the abdomen swollen, in the middle of the tumefaction was a circumscribed blue-red spot, the surrounding tissues being of a diffuse, bluish color. The skin, sub-cutis, and muscles of these parts were filled with a clouded, red fluid. A yellowish, serous exudate in abdominal cavity. The serosa of the stomach and intestines rose-red in color; * * * mucosa of small intestines but little swollen, that of large not at all. Spleen normal. Liver and kidneys clouded. * * * Inferior portion of both posterior lobes of the lungs bluish-red, dense, and resistant. Section of the lungs showed all parts to be red and œdematous." [In swine plague only the diseased parts of the lungs are œdematous!]

The second hog died on the 28th, or 48 hours after inoculation.

"Posterior extremities and inferior portion of the abdominal wall considerably swollen. The sub-cutis and muscles of the same infiltrated by a serous, hemorrhagic fluid. * * * Œdema pulmonum." Ibid., pp. 383-4.

No mention of lesions in the intestines. These are the essentials. Of these results, Schütz says:

"When we observe that Loeffler found a micro-organism in swine which corresponded in form and developed in the same manner with those cultivated by me, so far as can be judged by Loeffler's description [as 'Hülfsarbeiter' in the Riechs Gesundheits Amte, Schütz should know even more than that by personal observation!] we are justified in the conclusion that Loeffler's Schweineseuche is identical with the disease caused by the bacteria used in inoculating the above mentioned hogs, especially as the obduction showed almost the same changes as those recorded by Loeffler in the swine examined Oct. 26, 1882." Ibid., p. 385.

Here again we find all the essential phenomena of the "Wild-seuche," and none of the essentials of swine plague. Are we not, then, justified in asserting that the name "Schweineseuche"—swine plague—has been mistakenly applied by S. and L. to this German pest, and that all essential identity—phenomena between that disease and the true swine plague—are entirely wanting?

Is it not better that the Germans stick to their name, "Wild-seuche," for the disease they have so far investigated, and wait until they know

whether they have the swine plague proper before they use their term for expressing it, viz.: "Schweineseuche?"

The next question is

HAVE THE GERMANS THE TRUE SWINE PLAGUE?

It will be observed that neither Loeffler nor Schütz have thus far described any lesions in the lungs of the swine examined by them which even intimated the existence of broncho, caseous, or gangrenous pneumonia!

We will now see if we cannot find the direct evidence of still another swine plague in Germany that has not been differentiated by these leaders in original research.

SCHÜTZ'S CASES OF SWINE PLAGUE!?

"Case 1. Swine from Pülitz. Autopsy the 19th of September, 1885. Outside of stomach grey-red. Intestine bluish-red; the veins of the serosa and sub-serosa engorged with blood. Contents of small intestine fluid, that of the large intestine pultaceous; contents of the stomach contained a small quantity of cut up beets and some fluid strongly stained with gall. The mucosa of the regio œsophagea was swollen and gathered into deep rugæ, the greater part being deeply saturated with gall; the edge of the same bordering upon the lesser curvature was marked by two brownish-yellow spots of the size of a bean, which lay deeper than the surrounding tissue; these spots were sharply circumscribed, clouded, and dry, their edges could be easily lifted away from the surrounding mucosa. The glandular mucosa of the stomach was gathered in numerous folds, clouded, the combs of the rugæ were more or less strongly impregnated with gall; the spaces between these folds had an uneven surface and were of a greyish-yellow color. In the place of the lymph follicles were to be seen small excavations with smooth, flat edges and a bluish base. The mucosa of the duodenum and anterior portion of the jejunum was somewhat swollen; some few of the smaller vessels were partially engorged; further down the mucosa was not much swollen, and pale. No changes in the Peyer's plaques and solitary follicles. The mucosa of the cæcum and colon was clouded, bluish-grey in color, tending towards a green shade. The mesenterial lymph-glands were not excessively swollen, firm, and of a bluish-grey color. Spleen but slightly swollen. * * * Liver somewhat swollen, bluish-brown in color, and firm; edges somewhat rounded; the acini were distended, their central portion of dark brown color, the peripheral being clouded and greyish-brown. Gall-bladder partially filled with a thickish clouded gall; mucosa of a yellow color. Kidneys not much swollen,

grey-brown in color; surface smooth and refracting; the medullary portion had a yellowish-red color, while the cortical substance was greyish-brown, slightly clouded and marked by numerous engorged vessels. The pericardial sack contained 36 grammes of a clouded, reddish fluid. Peri and epicardium adhered together. When separated the adhesions were found to consist of elastic material; the separated surfaces were lustreless and covered with verrucous growths; the myocardium was of a greyish-brown color, and somewhat clouded, yet firm; the cut surface was dull. The lungs were large; both lobes of the left lung, with the exception of the superior edges, and the four lobes of the right lung were dense and atelectatic—hepatized. In the thoracic sack was 64 grammes of a clouded, reddish-yellow fluid containing flocculi of fibrin. The surface of the lungs was rough, clouded, and lustreless over the consolidated portions, which were of a general dark red color, with greyish-yellow and greyish-red centers scattered through it of various dimensions.” * * * Ibid., p. 391.

No mention is here made of “enormous œdema,” the balance of the description being merely a more detailed account of the lesions in the lungs. Following on the above autopsy are short notes of one upon a second pig, which offered no essential variation from the former. If we compare these necroscopical notes with those previously quoted, it would seem as if the merest novice in pathology would at once see that he had to do with cases offering manifestly different lesions than those described by Loeffler, and which Schütz concluded represented the same disease he had been investigating until we come to this point in his researches. That Schütz found himself somewhat in a quandary over these Pülitz cases is evident, for he says of them:

“These cases present much that is worthy of note. Both of the hogs presented the phenomena of an acute pleuro-pneumonia, which in hog No. 1 extended to the pericardium. The pneumonia had a malignant character. The yellowish districts, which were to be seen through the pleura, or complicated it, corresponded to necrotic portions of the lungs, and often destroyed a whole lobulus. I wish to call special attention to the small, yellowish centers which were described in the tissue of the lobuli, and which embraced the bronchioles. When we further consider that the dense, circumscribed centers in the soft parts of the lungs presented a reddish color, we are justified in concluding that the necrotic conditions have been the result of hemorrhagic inflammatory processes. The pneumonia of both swine is therefore a ‘multiplum’ of diseased centers, which present the appearance of following one upon another. Each of these yellow centers is the result of a local pneumonia, and had its individual beginning.

Consequently, the entire lung has been the seat of a multiple gangrenous pneumonia." Ibid., p. 392.

Had Schütz been more thoroughly competent as a "field" investigator, or less of a laboratory researcher, in fact, had he had a practical acquaintance with the diseases he was investigating upon cadavers alone, he would have also found something "Bemerkenswerth" in the clinical history of these swine, and which differs materially from the same history of his other swine, had he known anything about the practical side of the disease they died of.

Of this clinical history Schütz says, by this "careful and exact description!" If an American farmer could not do better than the following he would be a disgrace to the profession. Still, poor as it is, we can recognize the true swine plague in it, and easily see the points of clinical differentiation between the symptoms described and those given for the "Wild-seuche."

The story is as follows:

"After the newly purchased swine had been in the pens for three or four days, a diarrhœa set in. [It terminates the disease in "Wild-seuche," but, while also coming as one of the conclusive phenomena in swine plague, it also is one that first attracts attention.—B.] The discharges were of a greenish-yellow or greenish-grey color. [Swine plague!—B.] In rare cases they were hemorrhagic. At the same time the animals were stiff in their movements and lay down much. Appetite poor. In the course of the sixth or seventh day they became weak in the back and uncertain in their posterior movements (!) Weakness increased. By some (! ? B.) animals, at this time, the ears became red, as in Rothlauf; all had difficulty in respiration. The course of the disease was from eight to ten days." Ibid., p. 390.

What we miss at once here is the deformation of the "enormous œdema," which, had it been there, "Herr Kollege Hirschel" must certainly have noticed. He should also have known that neither the "Rothlauf" or "Wild-seuche" require "eight or ten days" to run their course.

After describing the phenomena in detail, Schütz proceeds to reason upon them, as follows:

"On this pneumonia follow the phenomena of a general infection, which occurs both by the lymphatic and circulatory systems. For the first speaks the severe changes in the neighboring lymph-glands, and for the infection of the blood the parenchymatous changes in the

liver, spleen, kidneys, and heart and the irritation of the stomach and intestines." *Ibid.*, p. 392.

One is surprised to see so good a pathological anatomist as Schütz fall into such a serious error as the above, especially one who sat for so many years at the feet of Virchow. Schütz has manifestly put "the cart before the horse," which he certainly would not have done had he had more experience. As a rule, these lesions follow the general infection, as is the case when pneumonia complicates typhus abdominalis, though when the infection takes place through the lungs a portion of these lesions may be concomitant and follow along with the specific septicæmia, but in such cases the broncho-pneumonia is the initiatory lesion in the lungs, while the gangrenous complication owes its genesis to the subsequent bacterial embolism, which is the direct result of the effects of the "general infection," septicæmia, upon the circulation.

Of the micro-organisms found in these pigs, Schütz says that he prepared numerous covering glasses from different parts of the lungs and proved them by examination.

"To my great astonishment" he says, [But why he should be astonished when he considered the disease to be the same that he had been previously engaged upon, is a question.—B.] "a great number of oval bacteria, of different sizes, were found in the hepatized portions of the lungs; many were exceedingly small, others somewhat larger, while still others were in the process of segmentation; the last showed every transitory stage between oval forms, with an uncolored place in their centers, to longer bodies, which, like the organisms of the *hen cholera*, only colored intensely at their ends, while about one-third of the body remained uncolored." *Ibid.*, p. 393.

Schütz, himself, seems to have been perfectly aware of the possibility of the very error into which he eventually fell, when he says:

"When the bacteria discovered in the lungs [in this case.—B.] and the spleen [in the former.—B.] correspond in form, it is not the logical conclusion that they are identical organisms."

But when, however, he found that they developed in identically the same manner, and that apparently similar results followed the inoculations of small animals, he at once jumped to the conclusion that it was the *Loeffler* organism again before him. But had he himself, or will he now, compare his own records, he will find that he describes

very much less swelling of the skin at the points inoculated, and that none of these latter animals presented the "enormous œdema" of the former, or so much tendency to local hemorrhages. Again, it would be well were he again to prove the action of these two similar organisms upon pigeons, for he might find that the immunity of those birds in the first case was not accidental, and that the fact that they did succumb in the second had some differentiating value.

To German investigators I would suggest the intra-abdominal injection of cultures from such cases as the above, instead of the intrapulmonal, as adopted by Schütz, when they come across this caseous broncho-pneumonia, and they can then settle the question as to the relation of these lesions to those described by Roloff, to which I shall refer shortly; but before doing so I must say that I do not place any extra value upon the diagnostic worth of the inoculation of small animals in differentiating these germs from one another, except where one species has a decided natural immunity to a given germ, or where one invariably produces some specific lesion, but the whole belted, ovoid group will produce septicæmia, if they produce anything, and the chief reason that caseous-gangrenous pneumonia results in swine plague, and has not been mentioned in the "Wild-seuche," probably is that the blood poisoning is far more rapid in the latter than the former, which also accounts for its greater hemorrhagic character. Again, there can be no doubt whatever that the micro-organisms of the "Wild-seuche" infect very easily through traumatic lesions of the cutis, hence the "enormous œdema," while hundreds of inoculative experiments show the same not to be the case in swine plague. Mr. Salmon and I agree on the latter point. That the hog by which Schütz inoculated two "Pravazsche Spritze voll" (!) of a culture, at one o'clock of the 16th, should die in the night of the 18th-19th of January, is by no means surprising, so we will pass by that experiment.

Allusion has already been made to the records of Prof. Roloff, and we will introduce the same by quoting another autopsy from Schütz, and then his opinion of the relation which the lesions described by Roloff bear to this disease. I wish it distinctly understood that it was the presence of these caseous-gangrenous lesions in the lungs of the latest swine examined by him that led Schütz to the idea that the intestinal lesions described by Roloff might possibly add to the com-

pletion of the patho-anatomical picture of the disease, as is illustrated by the following:

"Some animals do not succumb to the acute inflammation of the lungs, but in them there develops caseous conditions of those organs, the lymph-glands, and other organs, which extend in a progressive manner, strongly resembling the processes of tuberculosis, and finally lead to the death of the animal through general marasmus and loss of power.

"Cadaver much emaciated. Rigor mortis has disappeared. Visible mucosæ of a pale, bluish color. Right hind leg much swollen, the swelling beginning an hand-width above the patella, and extending below the hock. The surface of the skin was smooth, being only interrupted upon the anterior part of the metatarsus by several roundish nodes. Above these the skin was hard, like cicatricial tissue. In the midst of this dense tissue were many cavities, which contained a yellowish, caseous mass; the inside walls of the same were smooth. The joints and sheaths of the tendons were filled with a thick, mush-like material. Several of the bones of the tarsus were necrotic, and easily removed; others were corroded, and the seat of periosteal deposits. Upon the inner surface of the limbs were numerous noduli, which lay in the course of the lymphatics; they varied in size from that of a cherry to a walnut; some of these were solid, while others fluctuated; they were surrounded by a dense capsule; the contents were either of a yellowish-gray pultaceous material, or a desiccated, friable substance. The right inguinal gland was of the size of a hen's egg, its walls being thick and dense; contents semi-fluid; other glands of a similar character were found in different parts of the body.

"The right tonsil was much enlarged, and of a bluish-grey color; its pockets were filled with a viscid, greyish-white fluid. In the middle of the surface of this organ was seen a sharply circumscribed center, of the size of a ten-cent piece, which was of a yellowish color, and contained a caseous material; this object extended entirely through the body of the tonsil; on pressing out the contents, a smooth, walled cavity remained. The mucosa of the larynx contained several caeous ulcerations.

"In the thoracic cavity was 150 grammes of a clear, aqueous fluid. Lungs appeared large. The apices of both the anterior lobes and the inferior edges of the others were of a greyish-red color, atalectatic (airless), anæmic, and dense; the external surface of these portions of the lungs presented many retractions, and was adherent to the ribs in places.

"The balance of the pulmonary tissue was red, soft, and contained air. In all parts of the lungs were distributed numerous nodular neoplasms, varying in size from a millet seed to a walnut; those situated immediately under the pleura were of a dirty yellow color, and

projected above the general surface of the organ ; some had cavities in them, while others were firm and contained a caseous material. Many cavities connected directly with the ramifications of the air tubes. The walls of the cavities were smooth, their contents a soft, pultaceous, homogeneous mass of a yellowish-white color. Other nodes were surrounded by a dense capsule, their contents being desiccated and caseous. No fresh complications were to be seen. Bronchial lymph-glands as previously mentioned.

"Heart small, and of a pale brown color. Pericardial sack contained a table-spoonful of a clear fluid. In the abdominal cavity was found 250 grms. of a fluid as above. Peritoneum pale-grey; some vessels in omentum injected. Stomach contained a considerable quantity of a greyish-colored fluid. Gastric mucosa of a greyish-yellow color. Duodenum contained a yellowish-colored fluid. The intestines were so soft that they were scarcely to be separated from the mesentery without tearing. Mucosa not changed. Some of the lymph-glands of the mesentery presented conditions similar to those already described.

"These results reminded me very strongly of scrofulosis or tuberculosis, but in spite of every precaution and much labor I could not find a single bacillus of tuberculosis in the tissues ; instead of which I found the oval bacteria of swine plague. They were present in great numbers in all the caseous nodes. In size they corresponded with the younger generations which follow the rapid development of these organisms. In the necrotic caseous tissue, therefore, they developed in the same manner as outside the body under artificial conditions." *Ibid.*, pp. 408-9.

Cultures were made from this material, and apparently the germs found as in former cases.

Such chronic cases as the above would be hard to find in this part of the United States, where such swine would be soon burned up, before any one had an opportunity to examine them. I will also say that tuberculosis in swine is an unknown disease among the hogs of our western states, and is probably not a natural disease among these animals, as my observations in our eastern states have shown me that only swine getting the offal from beef slaughter houses, or dairy refuse, are afflicted with tuberculosis. Caseous processes in the lungs seem to be the natural product in swine, however, on severe and prolonged irritation of the parenchymatous tissue of the lungs.

LESIONS DESCRIBED BY ROLOFF* SHOW THAT THE GENUINE SWINE PLAGUE EXISTS IN GERMANY, AND, TAKEN IN CONNECTION WITH THOSE DESCRIBED IN THE SECOND SECTION OF SCHUTZ'S WORK, COMPLETE THE FULL PICTURE OF THE DISEASE.

“SCROFULOUS-CASEOUS-ENTERITIS.

“This chronic enteritis, or inflammation of the intestines, makes itself manifest in that young swine which have been perfectly well since their birth, and presented a fat and rotund form, gradually begin to emaciate; the skin becomes pale and uncleanly, and the appetite diminishes. To these phenomena diarrhœa sets in, being at first moderate, but gradually increasing in intensity; the discharges are very offensive. During the last weeks of life the animals become exceedingly emaciated, the back arched and backbone prominent, flanks fallen in, and abdomen tucked up, in its posterior parts, but pendulous in its lower parts, though not so much so as when ascites is present. The conjunctivæ are pale, the eyes sunken, heart-beats weak and accelerated. The appetite for solid food disappears, while the thirst increases.”

The professional reader, as well as swine breeder, who is acquainted with our swine plague, can easily see that the greater part of the above description applies very well to their experiences in that disease, but not nearly so much as that which follows:

“More exact examination of the abdomen, especially by pressure from the hands of the observer, allows one to feel the intestines, through the emaciated walls, which present themselves to the touch as a thick, dense mass, the individual convolutions not being discernible. The entire convolution of the large intestine is more like a large tumor, of about the size of two fists, its surface being marked by nodular or swollen irregularities. The examined animals frequently show pain on pressure.”

In cases where the diarrhœa is violent, death results in the course of a few days.

“NECROSCOPICAL OBSERVATIONS.

“The first thing that strikes the eye of the observer is the degree of emaciation and decrease in the amount of blood which the subject presents.

“The contents of the stomach are, in general, almost fluid in character. Many cases do not present any pathological disturbances in the stomach; in others the serosa of the great curvature presents

* Die Schwindzucht, fettige Degeneration, Scrophulose und Tuberculose bei Schweinen. 1875.

spots of diffuse redness, which correspond to dark or brown-red spots of irregular form in the mucosa of that organ, in which still smaller extravasations may be distinguished. The reddened portions of the mucosa are somewhat swollen, and often finely granulated superficially. Upon most of these places may be seen a pseudo-membranous covering, which on removal shows the underlying mucosa to be filled with small petechial extravasations.

"The mucosa of the duodenum is generally swollen, reddened, and uneven in the vicinity of the pylorus; that of the posterior portion and the balance of the small intestine is not so much swollen, but is full of small hemorrhagic centers, although of a general leaden-grey color. Other changes of importance are seldom met with in the small intestines. In seldom cases, however, when the ilco-cæcal valve is very severely diseased, the long Peyer's plaques in the vicinity are found swollen and the seat of small, round ulcerations.

"The mesenterial lymph-glands are more or less swollen, and generally (? B.) contain centers of cheesy degeneration.

"The pathological changes in the large intestines are, in all cases, very striking."

Roloff's description of them is, indeed, so striking, that I desire to call the most exact attention to them.

He proceeds as follows:

"The large intestines form a thick, dense packet, the single convolutions being united together as a conglomerate mass, the surface showing the regular markings of the individual folds more or less distinctly. Upon the surface of the individual convolutes are to be seen flat protuberances, some of which are roundish, while others are quite oval, varying in size from a five-cent piece to a quarter of a dollar; these neoplasms are surrounded by a slight ring of indurated tissue; upon these protuberances are also to be seen still smaller vesicular projections, varying in size from that of the head of a pin to that of a pea, or numerous small, less prominent, clouded points. Between the intestinal convolutions may be seen the swollen lymph-glands, which present an uneven surface to the eye of the observer. The external surface of the intestines show, on many convolutions, large, brownish spots, in which may be seen many small vessels which are distended in many places along their course, and are marked by numerous extravasations of varying extension. Other portions of the serosa present spots of diffuse redness, while others present a yellowish-red shade, the balance of the covering being quite pale. The serosa retains its normal lustre upon the slight reddish and pale portions, while it is clouded over the brown-red spots.

"By means of touch it is very easy to see that the increase in volume of the intestine is caused by a thickening of its walls, which at the same

time gives to them a certain degree of inelasticity. These conditions are most marked in the reddened portions. The secondary, flattish protuberances also feel dense, while the vesicular eruptions upon them fluctuate distinctly on pressure. The individual convolutions may be easily separated, as the mesentery uniting them is very friable. Between the convolutions in the markedly hyperæmic connective tissue are to be found the much hypertrophied lymph-glands, many of which have a medullary character while others have undergone caseous degeneration.

"The contents of the large intestine is, in general, represented by a small quantity of evil smelling, pultaceous or fluid stuff which is mixed with materials of a firmer consistency and is of a dirty, greenish or brownish color.

"The ileo-cæcal valve projects into the lumen of the cæcum as a long, dense, cylindrical body; its surface is of a leaden-grey color and disturbed by numerous openings of the size of a pin's head; its free extremity is marked by ulcerations which give it a very irregular appearance. The mucosa of the cæcum, in the vicinity of this valve, as well as along the colon, presents a very irregular surface, and is full of patches of a greyish-black color, and full of clefts which interrupt the continuity of its surface; between these greyish-black patches the mucosa is clouded, of a leaden color, its surface being also very uneven, while in other places it remains smooth and retains its normal lustre, but has a sort of granulated appearance. The round or oval neoplasms, previously alluded to, present a center having either a greyish-black or quite black color, their surface being clefted and irregular, while their peripheries are less dark, being of a dead grey color, the clefts and irregularities being less and less marked as one approaches the outside limits of these projections, until on their extreme limits they present a finely granulated appearance. The thickness of these objects increases from their peripheries towards the center. The clefted tissue is dry and friable in the center, but becomes more moist and consistent towards their circumferences, but still wants the tenacity of the normal mucosa. Sometimes we find clusters of these objects lying in close apposition to one another, or they become confluent, forming a mass lying either transverse or longitudinal to the course of the intestine. It is always easy to be seen that these spots are formed by the confluence of several of these round neoplasms, while their centers are marked by the darker and more clefted portions of the mass, or marked by loss of substance in the mucosa where desquamation has taken place. The seat of these changes corresponds exactly to the circumscribed and indurated portions of the serosa, previously described, which bear direct relation to the degree of disturbance which the inside of the intestine has undergone.

"All those places in the interior of the intestine which have the

above described clefted and granular appearance are surrounded by a wall-like elevation of the mucosa, which, in general, has the width of one's little finger. Between this wall and the neoplasm is to be seen a slight furrow encircling the same. Where two of these objects lie in close apposition, or any portion of them approach each other, this wall-like elevation of the mucosa is pushed in between them. These wall-like separations are either of a pale grey or yellowish-grey color, or else reddish; directly under their surface run a large number of distended blood-vessels, which have given rise to numerous extravasations; these surrounding tissues, again, are frequently of a very dark, bluish-red color. They feel tolerably hard, but are much less consistent than the normal mucosa.

"Between these changes one sees places of more or less extent where the walls have retained their normal thickness and have a delicate slate color. As a rule, one finds the solitary follicles much swollen in these parts. The pathological changes in the colon, while of the same nature, are generally less extensive than in the cæcum. In many places the villi, between these neoplasms, are of a blackish color, while in others the continuity of the mucosa has been interrupted by the breaking away of its substance. The pathological changes become less and less as one approaches the posterior end of the colon, although they frequently extend into the anterior portion of the rectum. The posterior part of this intestine is generally free from pathological changes other than an intensely swollen condition of the solitary follicles."

So much for Roloff's "chronic caseous enteritis." To the competent professional it must certainly appear as a very complete description of the gross lesions met with more or less frequently and looked upon as pathognomonic—characteristic—of our American swine plague. In fact, I challenge any one to show so complete and exact a description of those lesions in the publications of any American observer.

On plates IV. and V. will be found illustrations of the cæcum and colon of a hog, the autopsy of which has already been recorded, but a portion of which is here reproduced for the convenience of the reader in comparison.

This hog died in an outbreak of swine plague, Lincoln, Neb., October, 1886, from which I made a large number of autopsies and experiments.

Middle sized, black and white pig. No discoloration of cutis. Lymph-glands all swollen, and many of them a diffuse, dark, blue-

red color, others somewhat mottled in appearance. About one quart of a dark red fluid in abdominal cavity. Peritoneum covered with dark red spots of varying dimensions. The outside of the large intestine was of a dull grey color, clouded, swollen, and covered with a thick, viscid mass containing many flocculi. The mesentery itself was also swollen, its vessels being distended with a dark red fluid, and its surface covered with the same material. Several masses of a gelatinous character, having the color of Canada balsam, floated about in the abdominal effusion. The large intestine was attached loosely to the abdominal wall at three different points. These places were marked by being indurated and hard, their limits being sharply defined, and the vessels in the serosa over them being much more plain to be seen than in their immediate vicinity. The outside of the small intestine was of a dull greyish color.

Contents of large intestine semi-fluid. Mucosa somewhat swollen, and of a dull leaden color, and covered with a viscid adhesive coating.

Ileo-cæcal valve intensely swollen, indurated, and elongated, extending one and a half inches into the cavity of the intestine. Around its base, and extending for about two inches in all directions, was a pseudo-membraneous material of a coal-black color on its surface. The underlying tissues were indurated even to the serosa. The mass on top was dry and friable, but as one got deeper into it became more yellowish-grey, until it became little more than grey, anæmic, cicatricial tissue. About four inches from this enormously enlarged valve were four large, round, sharply outlined indurations, the centers of which were infundibuliform (that is, pitted) and of a black color. This center was surrounded by a mass, the surface of which grew lighter in color as it approached the outside limits. This mass was arranged in concentric layers, which gave to the whole surface of the productions a clefted appearance. These objects extended about one-quarter of an inch above the general level of the inside of the intestine. A patch of the same character, formed by the union of several similar objects, was situated about four inches deeper down the gut, and numerous isolated productions of similar character were distributed through the colon and a portion of the rectum.

Thoracic cavity:

Both sides contained a reddish-yellow fluid, in which floated masses of a honey-colored, gelatinous material. Lungs adherent, both sides;

anterior portion of both lungs solidified, œdematous; caseous bronchopneumonia, with formation of cavities.

It will not do for Salmon to tell me, as he has the public, that:

“Our (his) investigations have shown the existence of another bacterial disease in swine, which may be associated with hog cholera in the same herd and in the same animal.”

For I shall defy him and his tools by pronouncing that assertion unqualifiedly false.

It will not do for him to call on Schütz to support him in his endeavors to swindle the American public by forging a description of bacteria, which do not and never have existed except in the clouded intellects of the bureau of animal industry, for Schütz himself gives evidence that they probably belong to the true swine plague in Germany, and I have tried to show that Schütz has already described the balance of the lesions.

He says:

“In anticipation of future remarks, I will draw attention to a disease of swine described by Roloff under the name of “*Kasigen Darmentzündung*,” which he (Roloff) looked upon as a form of tuberculosis, but which, with the greatest probability, should belong in the group of diseases caused by the oval bacteria. Thereby we must not leave out of consideration the fact that the intestinal wall can be affected from the circulation, and consequently that the caseous condition of the intestines need not necessarily be attributed to the presence of the bacteria in the intestinal canal.” pp. 412, 413, *Arbeiten a. a. Kals. Gesundheitsamte*, 1886.

With this we close our discussion of the swine plagues of Germany, and feel very strongly convinced that future investigation will demonstrate that the Germans are even more blessed with porcine pests than we are, having three, though the three together do not do as much damage as our one, from the fact that the chances for land infection (Mr. Salmon!), or better, exposure upon infected lands, is less in Germany than here in America, on account of the half-open pens and yards of the West, and hence extension over the country is less restricted also.

These three German swine pests are:

1. Rouget, or erysipelas, limited to swine, according to the literature.

2. "Wild-seuche" (Loeffler-Schütz "Schweineseuche"), infects deer, cattle, and swine.

3. Swine plague, limited to swine, and, as far as the literature is concerned, is made up of Schütz's "infectious pneumonia," and Roloff's cascous enteritis.

The burden of evidence to the contrary now rests upon the German investigators to furnish.

SWINE PLAGUE IN SWEDEN AND DENMARK.*

"A very malignant and infectious disease has prevailed among the swine of Sweden and Denmark since last fall (1887)."—Selander.

Prof. Bang, of the Veterinary School at Copenhagen, gives the only accessible (to me) description of the disease in hogs, and unfortunately that is very imperfect, and hence unsatisfactory. He says (*Wochenschrift*, 1887):

"This pest is neither identical with the disease known as 'Rothlauf' nor with the 'Schweineseuche' of Germany, but more like the American hog cholera or the swine plague of England." [Which, as has been shown, are one and the same disease; that is, using the term "hog cholera" in its generally accepted sense.—B.]

Bang continues:

"Young animals are the most frequently infected, and succumb the easiest to the disease. The diseased animals become weak, somnolent, and frequently stupid, and can only be moved with difficulty. The temperature is increased to 41° C. Appetite ceases; the alvine discharges are at first retarded, but soon become catarrhal and very fluid, having a very disagreeable odor, and are sometimes mixed with blood. The eyes are dull, the lids being frequently agglutinated together by a dirty secretion. The respiration is difficult, and voice hoarse. The animals become much emaciated during the course of the disease; they also become very weak, especially in the posterior extremities; many have more or less redness of the ears, limbs, and abdomen. The tonsils are often the seat of diphtheritic complications, if the mouth is subjected to an examination. On autopsical examination the contents of the large intestine are generally found to be thin and of an offensive odor; the mucosa is inflamed or swollen, reddened, and covered with a greyish-yellow coating."

Dr. Selander, "Ueber die Bakterien der Schweinepest." *Centralblatt für Bakteriologie und Parasitenkunde*, Vol. III., p. 361, 1888, and *Wochenschrift für Thierheilkunde*, 1887, p. 453, and 1888, p. 78.

The identical resemblance to many cases of American swine plague is so easily to be seen that an ordinary breeder would recognize the diseases to be the same, yet it is to be regretted that the autopsy notes are so meagre, for they show an insufficient knowledge of the disease in its ramified representations.

What kind of a micro-organism have they found in this disease?

We will now turn again to Selander, who says:

"As this (Swedish) disease has been thought to be identical with the German 'Schweineseuche,' and the German government quarantined against swine from Sweden and Denmark, Dr. Bang went to Berlin to see if he could not get it raised. He took cultures of the micro-organism of the swine pest (Swedish) with him, in order to show the identity of the two diseases. These cultures were turned over to me. On my return to Sweden I have convinced myself that the Swedish swine pest is caused by bacteria which are identical with those taken to Berlin by Bang, but have nothing to do with those of the German Schweineseuche."

Now how about these bacteria?

"When inoculated in gelatine they develop punctiform colonies in the course of two or three days; they develop slowly, the growth extending but slightly above the surface of the gelatine, which they do not cause to become fluid; * * * they grow upon potatoes, and have motion; in gelatine cultures they vary from a somewhat bacillary to almost a coccus form. In the tissues they always have the form of bacilli. (!?—B.) They are difficult to color with the ordinary aniline, and often have a spore-like, clear space in the center of their bodies, but do not develop spores. They color best in alkaline methyl blue solution, carbolized fuchsine solution, and by Kuhne's method." pp. 362-3.

The above are the essentials of Selander's description, which, meagre as it is, is sufficient to show its exact correspondence with the germ described by me in former pages. More experience will show Selander that these organisms develop in the same manner as they do in fluid cultures, showing their ovoid, belted, or elongated, belted, or ovoid, coccoid forms in tissues which offer little resistance, but that they develop not only in a seeming rod form, but also in long threads, when in delicate capillaries, running between more or less consistent tissues, but even here the uncolored middle space may be distinctly seen if the tinting and after treatment have been proper and one knows how to see them. It would have been well if Selander had told us how

these organisms differ from the Loeffler microbe when such difference is so marked as to justify him in saying, that they "have nothing to do with the organisms of the German Schweinesenche." That the microbes of "Wild-seuche"—the Loeffler disease—do grow upon potatoes, is assured by both Hueppe and Kitt, and that those of the American swine plague will, also, is assured by my never having failed in making them, and that the Cornil-Chantemesse disease is the same as the American is shown by their report, as well as that their organism also grows upon potatoes, which is again the case with that of Reitsch and his confrères, who also describe the same disease, so far as one can judge from their report;* and as I have shown that Schütz also had to do with the same disease, we may feel assured that that organism will also grow upon potatoes. As to the motility question, as I have shown elsewhere, the dispute lies upon what may be looked upon as independent movement, "*Selbständige Bewegung*," and not as to whether they move or not. That these germs move, and that it is not the "Brownian movement," is, to my mind, clear.

As to growth in other directions, I can see no differentiation of sufficient value to call such in the description of Selander from that of Loeffler, and yet, with him, I should insist that the germs were not identical, because their pathogenetic results were so dissimilar.

From the foregoing it will be readily seen that these Swedish and Danish observers are themselves of the opinion that the pest destroying their hogs is identical with that of England and the United States, and I have conclusively shown that the two latter are one and the same disease, both by the lesions which occur in the diseased hogs, and by the germs which are to be demonstrated in those same tissues. Furthermore, we have the evidence, given by these observers, that "the disease was imported into Sweden by breeding boars from England." *Wochenschrift*, l. c. 1888, p. 78. This being so, we have the evidence from Klein's illustrations, Plate II., Figs. 3, 4, and my own confirmatory demonstrations of the germ, that the micro-organism which causes the disease in Sweden and Denmark must of necessity be identical with that of Detmers and myself in this country, and Cornil, Chantemesse, and Reitsch in France. It is to be regretted that Selander did not find opportunity to make some observations upon Schütz's infectious pneumonia, both in the laboratory and the field.

* Recently confirmed by Cornil and Chantemesse.

SWINE PLAGUE IN FRANCE.

Ever since I began my investigations of this disease in Nebraska I have been surprised that we had no account of its existence in France. There is no question, in my mind, that the disease has existed in France for centuries, but that, as in Germany, it had been confounded with the "Rouget," and probably looked upon as a chronic or abortive form of that disease. The stimulus to the study of the diseases of swine, which was inaugurated by Schütz, when he demonstrated that there was another porcine pest in Germany than the "Rothlauf" ("Rouget," French), probably extended to France, and we may not therefore be surprised at the fact of the discovery of a second porcine pest in that country. This discovery was made by Cornil and Chantemesse, the former being the most able pathologist in France. This account is to be found in the "*Comptes Rendus*," Dec., 1887, p. 1281, under the following title: "*Etiologie de la pneumonie contagieuse des porcs*," by MM. Cornil et Chantemesse, and is as follows:

"There has existed in France for several years a disease of hogs which is contagious [! ! ?—B.] to an extreme degree, and which terminates in a fibrinous pneumonia [caseous ?—B.], and generally terminates fatally. The malady has not (previously) been recognized, having been confounded with the "Rouget," but the system of inoculation which has been found to be advantageous in the latter has no value against the former disease.

"This contagious pneumonia was first observed towards the end of the year 1883, in the herds of swine in the vicinity of Gentilly. Little by little the swine breeders were ruined by their losses. Every effort to control it seemed fruitless. The diseased animals at first appear weak, and seek secluded spots, where they lie down. At the same time a cough makes its appearance, accompanied by much difficulty in breathing. The appetite diminishes and the animals become emaciated. The skin of the abdomen and inside sometimes assumes a reddish hue, which has been the cause of the disease having been mistaken for rouget. The animals lie motionless, and express pain if forced to move. A mucous diarrhœa is present from the beginning [! ! ?—B.], the discharges having a disagreeable odor, and sometimes continues to the termination of the disease; at other times the animals are constipated. The course of the disease extends from twenty to thirty days, which decidedly differentiates it from the rouget, as well as do the pulmonary complications and the character of the micro-organism connected with it.

"We had previously found small bacteria in the sections of por-

tions of diseased lungs which had been placed at our disposal by M. Meguin; when in June, 1887, a gentleman of Gentilly begged us to investigate a disease from the effects of which all his hogs were dying, and which had existed in his pens for over three years, in spite of the isolation of the sick animals and the cleaning and disinfection of his premises. [Those conditions are by no means conformable to a contagious disease, which such treatment should have entirely eradicated. —B.] * * * At our request the owner killed a pig that had been ill for several days, and which showed in its lungs the initial lesions of broncho-pneumonia.

"Sections were made from the lungs, liver, and spleen, as well as specimens prepared from the blood. In the two latter nothing was found. From the lungs and liver we obtained pure cultures, which presented the following characteristics: The organism which developed did not liquefy gelatine, but grew upon the surface as a transparent spot, sometimes thick, and at others spread over the surface of the material. They grew as milk-like colonies upon agar-agar. Upon potatoes the colonies had a greyish color. All these cultivations were made up of the same microbe. It is a small, ovoid bacterium, having rounded ends. It is not motile. [!?!—B.] Two pigs were inoculated from pure bouillon cultures of this organism, as well as some guinea-pigs, rabbits, mice, and pigeons. On July 1, 1887, we inoculated a pig into the substance of the right lung, with one-fourth of a gramme of a fresh bouillon culture, injecting the material with a hypodermic syringe. July 2, the animal showed signs of illness, ate but little, and remained in a recumbent position. The temperature was 40° C. Later on the animal had the diarrhoea, grew thin, and displayed increased and difficult respiration. At the locus inoculationis râles could be distinguished in the lungs, and as well as crepitations, which could not be discerned upon the other side. The animal died July 28. The necroscopical examination demonstrated the existence of a general broncho-pneumonia in the right lung, while but a few lobules were complicated in the left. The kidneys presented the conditions of an intense nephritis; the urine contained albumen. The large intestine was marked by the presence of numbers of solid tumors of various dimensions. The lymph-glands were swollen. In the exudation from the cut surface of the diseased lungs, in the lymph-glands, the liver, spleen, kidneys, urine, bile, and blood, were found the same microbes, in a pure condition, that were in the fluid used for inoculation. It was also found in abundance in the fæces. The same cultures also killed rabbits, mice, and guinea-pigs, but were innocuous in pigeons. The microbe was found to be very abundant in the blood of the mice, where it appeared to be somewhat larger, and shows a clear space in its center when colored with methyl-blue.

"The lesions above described show that the contagious pneumonia of swine is a general infectious disease, rather than a local pulmonary affection. The predominance of the pulmonary lesions shows that the virus acts directly through the respiratory organs, but the germs may also gain access by the digestive tract, or by trauma in the skin.

"This disease appears to be identical with that described by Schütz and Loeffler, in Germany, under the name of "Schweineseuche," as well as the one described by Salmon, in America, as a new disease, (?) under the name of swine plague, during the last year. [The Frenchmen seem to know nothing of Salmon's micrococcus and his nondescript swine plague germ of 1885.] The German observers have demonstrated the presence of the ovoid bacterium, as well as its pathogenetic action in certain animals.

"In our next communication we shall demonstrate the principal biological characteristics of this organism, as well as the process by which we have produced the attenuated virus by which immunity is secured."

It will be plainly seen that the above description corresponds to the lesions of swine plague in this country, and not to that of Loeffler and Schütz, except the second series of observations of the latter, and then only when Roloff's observations are included, but what is of especial value, and to which I shall again refer, is the fact that though pneumonia resulted from the direct inoculation of pure cultures into the lung directly, that "the large intestine was marked by the presence of numbers of large tumors," and that the authors should also have found Mr. Salmon's forged germ of swine plague in such a case.

A SECOND COMMUNICATION UPON SWINE PLAGUE IN FRANCE.*

"An epidemic never before recognized broke out among the swine of Marseilles and its suburbs in July, 1887. The general opinion seems to be that it was imported from Oran by swine unloaded at this place on the 25th of June, the first cases in the African hogs having been on July 10th. We began our investigations the latter part of August. Intestinal disturbances seem to be the prevailing complication. The disease, generally, terminated fatally in from ten to twelve days after the first symptoms appeared; in some cases its course extended over but three or four days. Diarrhoea is present in some individuals, while constipation prevails in others. Fever is not at all con-

*L'épidémie des porcs à Marseille en 1887. Note de MM. Rietsch, Jobert, et Martinand.—*Comptes Rendus*, Janvier, 1888, No. 4, p. 296.

stant. [Sic!—B.] The animals coughed but little. Weakness of posterior parts, with wabbling gait, common. Skin marked by localized red patches on the body and extremities. Old hogs not so susceptible as younger ones. The African swine resist the disease better than native animals. [Sic!—B.]

“Necroscopical observations frequently show the kidneys, liver, and lungs to be perfectly healthy; (à l'autopsie, il n'est point rare de trouver les reins, le foie, le rate, et même les poumons d'apparence tout à fait saine.) [I do not hesitate to condemn the above assertion as absolutely erroneous, with regard to the kidneys and liver, and showing an equally absolute ignorance of pathological anatomy, which in fact characterizes this whole communication.—B.] Spots [of what character?] are frequently seen upon the liver, as well as characteristic [how?] hemorrhagic centers upon the kidneys. When the disease has been of long duration the digestive tube is the seat of serious and characteristic lesions. The walls of the stomach are ulcerated and the small intestine is punctured ('l'intestine grêle est piqué'), or perhaps spotted is meant. [Puncture of the small intestine is not a lesion of swine plague.—B.] The surface of the ileo-cæcal valve is frequently ulcerated, which extends to the Peyer's plaques, which are the point of departure [!?!—B.] The cæcum and colon are the frequent seat of ulcerations, which often have a radial character, and are of a greyish color and are friable; they frequently lead to contraction of the lumen of the intestine. The tissues around them frequently lose their normal characteristics. The development of the ulcerations is to be easily followed in the rectum; the mucosa being swollen and follicles closed. In the cadavers of the swine examined by us, we did not find any trace of glandular structure at these ulcerated points, but the cavities of the follicles, or the space occupied by them, was filled with a white, caseous material, containing the bacilli. The muscularis is also somewhat complicated. Swine that have been ill some time often display ulcerations of the inferior surface of the tongue and the buccal membrane. The reaction of the stomach and intestines is always alkaline. Cultures from the spleen, kidneys, and liver were seldom followed by the development of any micro-organism. [Something wrong in the manipulation, as I have never failed in obtaining them from either of these organs, though the spleen has been invariably the one organ to which we always turned to get cultivations.—B.] The pulp of these organs only gave negative results when introduced into the sub-cutis of mice and rabbits. [Wrong again!—B.] More positive results were obtained from the pulmonary tissue. Still better results were obtained with the mucosa of the intestines and fragments from the ulcerations, the same bacillus being obtained in a pure or nearly pure condition. Analogous results have been produced from the lungs, rarely from the liver. This bacillus is motile!! (A ba-

eille n'est point immobile.) [This confirms a point which I have always insisted upon against other European observers.—B.] Death resulted from the inoculation of pure cultivation upon mice in about ten days. Rabbits do not seem to be affected by it. [A mistake somewhere!!—B.] * * * The character of the cultures corresponded more or less to the descriptions of the micro-organism of the German "Schweinesenehe," as furnished by Loeffler and Schütz, as far as can be judged. In some respects striking differences were observed!! [Why not tell what they are in a matter of such importance!?!—B.] On the contrary we were struck with the similarity of the lesions with those described by Salmon for the American hog cholera. Thanks to the kindness of Mr. Salmon, we were enabled to compare the two microbes.

"Our examination of the same in hanging drops, gelatine cultivations and upon potatoes, does not permit us to identify these organisms."

Here are several very important points for the reader to remember:

1. The germ of these observers corresponds in many particulars, and especially in the lesions produced, with that of Detmers and myself.

2. It did not correspond with the culture sent by Mr. Salmon as the germ of his hog cholera. (It would be most interesting to know what kind of an object Mr. S. sent over to France, and it is to be lamented that these observers are so wanting in going into details.)

3. Their observation, negatively asserting that their organism does grow on potatoes, confirms that of Cornil and Chantemesse, which in their turn confirms my own statement.

"Cornil and Chantemesse have recently studied (*Comptes Rendus*, Dec., 1887. See the previous article) a contagious pneumonia of swine in the vicinity of Paris, as well as these outbreaks at Marseilles. They pronounce the disease to be the same in both cases.* They attribute the variation of the symptoms to the different ways by which the virus enters the animal body. We have lately observed a number of cases in which pneumonia predominated in the eruption at Marseilles!! Pneumonic lesions appear to be the exception in the warm months."

I think it has been sufficiently shown that the observations of Loeffler, and partially those of Schutz, can never be confirmed by relying upon the micro-morphology or biology of these germs. It is the result, the lesions, studied among a large number of swine and

* See *Comptes Rendus*, Tome cvi., p. 612, l. c., where this assertion is again confirmed.

extended over a large extent of territory, which can only lead to a positive differentiation in this matter. It is for the Germans, now, to confirm the observations of the rest of the world as to the swine plague proper, and for us to see if and where we have the "Wild-seuche," and not for us to go vainly hunting after bacterial points of identification and constantly neglecting to appreciate equally trustworthy points, the clinical and patho-anatomical.

SALMON'S "HOG CHOLERA."

ARE THERE TWO SWINE PLAGUES KNOWN IN THE UNITED STATES
AT PRESENT?

It would seem as if it were superfluous and almost an imposition upon the reader to revert to this question again, but the latest communication from Mr. Salmon, to which allusion has already been made in our consideration of the etiology of this disease, renders it absolutely necessary in this place, in order that truth and right may prevail. Science is but the search after truth, after facts, and he who dodges the point, or he who, through a false ambition, falsifies or endeavors to mystify the public, is utterly unworthy of public confidence and the respect of honorable members of an honorable profession. I wish it to be distinctly understood that the strikingly polemic character of this work is not for any desire for notoriety, but in order to place this question of the specific cause and nature of the American swine plague so acutely before the American live stock growers, as well as the scientific world, that it must receive its early and positive settlement, for no one more fully realizes than I do, the injury to our porcine interests that is constantly accruing through these contradictions between myself and the authorities at Washington. Hence, confident in the general correctness of my own work and conclusions, and with no other purpose in mind than faithful service to my country, and sensitively jealous of my reputation as an investigator who sometime hopes to be recognized as a scientist, no other course is open to me, but to stand manfully by what I know to be the truth. The reader will certainly be just enough to observe that in quoting evidence I have endeavored, as far as possible, even at the risk of profuseness, to give the whole story and not to select passages from the works of others which were especially suited to support my own conclusions. The one thing I have sought to do is to get at the truth and place it honestly before the public.

But to the question :

ARE THERE TWO SWINE PLAGUES IN THE UNITED STATES?

At this place we shall endeavor to still further fortify our position that there are not, by referring especially to the lesions of the disease, in support of what has been previously noted, although the bacteria must also come again into consideration.

It will be remembered that Mr. Salmon opened his latest report upon this subject as follows :

“INVESTIGATIONS OF SWINE DISEASES.

“In view of the results of investigations which have shown the existence of two distinct infectious diseases in swine, perhaps of equal virulence and distribution, a change in the nomenclature becomes necessary, in order to avoid any confusion in the future. Since these two diseases have been considered as one in the past, and the names swine plague and hog cholera have been applied indiscriminately, we prefer to retain both names, with a more restricted meaning, using the name hog cholera for the disease described in the last report as swine plague, which is produced by a motile bacterium, and applying the name swine plague to the other disease, the chief seat of which is in the lungs. This change is the more desirable since recent investigations have shown that the latter disease exists in Germany, where it is called swine plague (*Sehweineseuche*).”—l. c., 1886, p. 603.

Under the title, “How can Hog Cholera be Prevented?” he tells us exactly what he means by “hog cholera,” when he says :

“Of this disease, the only reliable diagnostic lesion is ulceration of the large intestine, or the presence of the bacterium in the body of the infected animal.”—l. c., p. 654.

I pronounce that whole assertion as unequivocally false, and am willing that my scientific reputation shall stand upon the results in the future.

Again, Mr. Salmon says :

“Our investigations have already shown the existence of another bacterial disease in swine, which may even be associated with hog cholera in the same herd and in the same animal. From the present stand-point of our information it would be presumably absurd to rely upon *post-mortem* examinations in different parts of the country without at the same time making bacteriological investigations, in order to decide the nature of a certain class of symptoms and lesions. We have almost invariably found severe intestinal lesions in hog cholera, producing ulceration and often complete death of the mucous mem-

brane of the large intestine, involving in the severest cases a similar destruction of the mucous membrane of the ileum.

"In hog cholera, lung lesions are quite secondary, and only rarely seen. In the severest hemorrhagic type of the disease we have almost always observed hemorrhagic foci scattered through the lung tissue, but these were no more numerous or more extensive than the extravasations found in most of the other viscera. It is quite easy to believe that such cases surviving the first severe attack may develop a pneumonia by the gradual extension and confluence of the separate foci. In all cases where the causation of such lesions is a matter of doubt bacteriological investigation must now decide." U. S. Agricultural Report, 1886, p. 68.

Wherever one touches the statements of this author we always fall upon the most absurd contradictions. Above he has told us that, "the only reliable diagnostic lesion is ulceration of the intestines." We should be able to rely upon such a statement, but when we read the second quotation we find Mr. Salmon saying: "We have almost invariably found severe intestinal lesions in hog cholera producing ulceration," while in the next passage he tells about "lung lesions," and we shall show that he describes "lung lesions," and produces them by inoculation as frequently as he does "the only reliable diagnostic lesion, ulceration of the intestines." As to his microbe, I deny its existence in any known disease of American swine, *in toto*.

Will the reader have the kindness to most carefully compare the following quotations from Mr. Salmon's latest communication with the previous ones:

"Ulcers of the large intestine were found in 36 out of 49 cases, or about 75 per cent. They vary from very slight to very severe and extensive lesions, involving in a small number nearly the whole of the membrane of the cæcum and colon. In a few cases the lesion was not limited to the mucous membrane, but extended into the muscular wall, producing considerable local inflammation and thickening of the serous membrane. In rare cases necrosis and cellular infiltration had made the intestinal wall so friable that it broke when handled. In five cases the lower ilium was ulcerated, but the ulcers seemed to have no relation to Peyer's patches. The age of the ulcers cannot be determined, as the process of necrosis and ulceration seems to vary a great deal in rapidity. We have frequently found a combination of old ulcers with hemorrhagic lesions. These would appear to resemble the condition found in chronic pleuro-pneumonia, where we find a cyst and at the same time a fresh inflammation of the lung tissue. What conclusion is the pathologist to draw from this condition? Is

it the result of an increase of virulence of the bacteria which have been preserved so that they are able to penetrate and multiply in the tissues which have previously resisted them, or is it simply the result of an extension of the bacteria, which have not increased in virulence, but which have been limited to the affected part of an organ? The answer to these questions must have great influence on our views of the preservation of virus and the sudden appearance of virulent epizootics which apparently have their starting point in mild chronic cases.

"Peritonitis, pleuritis, and pericarditis are not uncommon complications usually accompanying old ulcers. These are possibly caused by septic bacteria gaining entrance through the ulcers. We have demonstrated that cocci closely resembling those of suppuration, and various other microbes, are usually found in the peritoneal cavities in chronic cases.

"The lesions found in the lungs on *post-mortem* examination were either simple collapse or lobular broncho-pneumonia, which apparently followed it. In about fifteen per cent of the animals examined one of the smaller ventral lobes was airless throughout and moderately enlarged. Viewed from the surface the diseased lobe is bright red, dotted with minute, pale, grayish and yellow points of a diffused, hazy outline, each not more than one millimeter in diameter. They are usually ranged in groups of four, and represent the ultimate air-cells filled with cellular exudate. The larger bronchi are also occluded. Microscopic sections reveal a broncho-pneumonia. The process seems to be accompanied with very little inflammation. The desquamation and proliferation go on in the alveoli and smallest air tubes until they are occluded by the casts. Of the forty-nine animals of the herd mentioned, seventeen were found with collapse and eight with broncho-pneumonia. In this outbreak, then, more than one-half of the animals had some defect of the lungs." *Am. Vet. Review*, April, 1888, pp. 8, 9.

"Seventy-five per cent" of "ulcers in the large intestine" cannot, then, be considered as "the only reliable diagnostic lesion," when we find also that "of forty-nine animals seventeen were found with collapse and eight with broncho-pneumonia, "and more than half of the animals had some defect of the lungs."

"Microscopical specimens revealed a broncho-pneumonia"!

Whoever heard of any competent patho-anatomist having to resort to a microscope to diagnose "broncho-pneumonia?"

Truer evidence of bureaucratic-Washingtonian myopia could not be desired.

We have, however, some more evidence of the same kind. It is as follows:

"There is an idea among some investigators that the lung lesions of infectious pneumonia or swine plague and the bowel lesions of hog cholera are produced by the same bacteria, and that the seat of the disease depends entirely upon the organ through which the germ is introduced into the body. To illustrate this matter we have injected the germs of hog cholera directly into the lung tissue, *but we have not produced hepatization* by such inoculations. Either the germ is diffused through the body, producing lesions of the spleen, lymphatics, and intestines, or the animals recover. *We have found, however, that in the lung lesions accompanying hog cholera there are a much greater number of germs than in the healthy portions of the lungs.* It is evident, therefore, that in these collapsed portions the germs find favorable conditions for their multiplication, and it is not unlikely that the collapse develops into broncho-pneumonia because of their multiplication. To determine whether the microbe of contagious pneumonia was present in the affected portions of the lungs from animals affected with hog cholera, sixteen rabbits were inoculated from the same number of lungs. Of these, eight lungs were affected with simple collapse and eight with broncho-pneumonia. Of these sixteen rabbits, four survived and the remainder died of hog cholera. The germ of contagious pneumonia or swine plague evidently was not present in any one of these cases." l. c., p. 10.

Will Mr. Salmon have the kindness to equalize the above contradictions?

1st. He tells us that, "we have not produced hepatization by such inoculations."

2d. That "in the lung lesions accompanying hog cholera there are a much greater number of germs than in the healthy portions of the lungs," and "that it is not unlikely that the collapse develops into broncho-pneumonia because of their multiplication."

Mr. Salmon, what is "broncho-pneumonia," but localized hepatization?

You tell us that "sixteen rabbits were inoculated from the same number of lungs," and that of these, "eight lungs were affected with simple collapse and eight with broncho-pneumonia;" that is, localized hepatization.

You also tell us that these sixteen rabbits were inoculated with, or from, "affected portions of the lungs from animals affected with hog cholera," and then, again, say, "the germ of contagious pneumonia, or swine plague, was not present in any of these cases."

Mr. Salmon, what germ then caused "the affected portions of the lungs of animals affected with hog cholera?"

What germ caused the "eight cases of broncho-pneumonia" in your rabbits?

"Evidently" the germ of "hog cholera" must have been present in these cases. "Evidently" it must have caused the "affected portions of the lungs" in the first case, and in the "eight (rabbits) with broncho-pneumonia" in the second. "Evidently" it causes "hepatization." What, then, in the name of common sense, is the difference in its action from the swine plague germ?

Again, Mr. Salmon, you tell us that "sixteen rabbits were inoculated," and that of these, "eight lungs were affected with simple collapse, and eight with broncho-pneumonia."

Eight and eight make sixteen, do they not?

Then how in the name of common sense and mathematical exactness could you have told your very intelligent audience that, "of these sixteen rabbits, four survived and the remainder died of hog cholera," when you had previously said that, "of these (sixteen) eight lungs were affected with collapse and eight with broncho-pneumonia"?

How did you find that out?

How did you discover that the germ of contagious pneumonia or swine plague evidently was not present in the "four survived" rabbits?

Do please answer, Mr. Salmon.

Did you guess at it?

Somehow, we have learned, or perhaps have been taught, that the basis of such a statement as the above could not be truly ascertained except by necroscopical examination, and unless we are very ignorant, such knowledge can only be gained by examining dead animals. Then please tell the world how "four" could have "survived," and the "remainder have died of hog cholera," when, according to your own statement, the whole sixteen must have died of something.

It will be shown that Prof. Wm. Welch, pathologist of the Johns Hopkins University, also found the same ovoid, belted germ, or, as he says, one like the Loeffler-Schütz organism, in a local outbreak of swine plague near Baltimore.

This comes very near home, Mr. Salmon!

But the reader will also observe that Mr. Salmon admits that pneumonia does occur in his "hog cholera," in the passage where he says:

"It is quite easy to believe that such cases surviving the first severe attack may develop a pneumonia by the gradual extension and confluence of the separate foci ;"

which, as has been shown, is just what takes place in swine plague, but not always by the "confluence of the foci," the pneumonia being invariably either a secondary or concomitant complication with the blood poisoning, and dependent upon the atrium by which the inficiens gains entrance to the infected animal, or upon the virulence of the germs and the effect of the septicæmia upon the circulation.

What then has Mr. Salmon left to stand upon?

Nothing but the germ of his own creation!

Let us now again recall that wonderfully conclusive and not to be contradicted testimony of Cornil and Chantemesse:

"On July 1, 1887, we (they) inoculated a pig into the substance of the right lung, with one-fourth of a drachm of a fresh bouillon culture. [It will be remembered that we have already shown that the micro-organism discovered by these observers was identical with our own, even to growing on potatoes.] July 2d the animal showed signs of illness. * * * Later on it had diarrhœa, grew thin, etc. * * * Died July 28th. The necroscopical examination demonstrated the existence of a general broncho-pneumonia. * * * The large intestine was marked by the presence of numbers of solid tumors of various dimensions. *

* * * In the exudation from the cut surface of the diseased lungs, in the lymph-glands, the liver, the spleen, the kidneys, the urine, and the blood, were found the same microbes, in a pure condition, that were in the fluid used for inoculation."

What testimony could be more conclusive? Even Mr. Salmon, in the address noticed, credits M. Cornil as an undoubted authority when he quotes him to show that his (Salmon's) "hog cholera microbe" had not been found in their work in France.

Will Mr. Salmon please tell the scientific world why, as an honest investigator should, he did not also tell those poor deluded veterinary colleagues of his of the above testimony of Cornil against himself?

He dare not! He knew full well that he was posing as an impostor before a body of half educated men utterly incapable of giving judgment upon his assertions.

When he quoted M. Rietsch and his colleagues, of Marseilles, as unable to find that same H. C. imposition of his, why did he not also quote what they said about his microbe, viz.:?

"Our examination of the same, in hanging drops, gelatine cultivations, and upon potatoes, do not permit us to identify the organism" with that discovered by them, which "corresponds more or less with that of Schütz and Loeffler;" nor does Mr. Salmon tell us that these observers also said that, "we have lately observed a number of cases in which pneumonia predominated in the eruption at Marseilles," although they also observed that, "when the disease has been of long duration the digestive tube is the seat of serious and characteristic lesions."

We have already shown that it was only when the work of Professor Schütz and myself had demonstrated Mr. Salmon's "micrococcus" (1880 to 1885) to have been the creation of a diseased imagination, that he manufactured this nondescript as the cause of swine plague, and when he found that he could not upset my work, and in order to try and save his reputation, that he created a new disease, "hog cholera," for that nondescript, in 1886.

We have also shown that, up to 1886, Mr. Salmon knew of but one swine plague in this country. We must be pardoned for again calling attention to that testimony. This nail of truth must be driven home and clinched once for all.

In 1878 Mr. Salmon said:

"We may estimate the loss (of swine) in the entire state (North Carolina) at about 260,000, for the year ending April 1, 1878.

* * In each of the counties named a considerable number of herds were visited and examined, and without exception the living animals presented similar symptoms and the dead ones showed similar changes in the different organs of the body. Slight variations were of course observed, as is always the case in any disease, but these were as great between individuals of the same herd, sick at the same time, as between different herds, even in different counties. And, what is of great importance, I did not find a single case in which it could possibly be supposed that death resulted from a local disease (!) but in every case a variety of organs, belonging to a different apparatus, were found diseased; the blood often showed marked changes; there was extravasations in various parts of the body, and always inflammation of the lungs and large intestines; generally, also, of the heart, and often of the eyes; the skin, too, was plainly affected, and the temperature was found to be increased before any other symptoms of disease were found to be present.

"Considering all these facts, there can be no doubt that these animals all died of a general disease, a disease not caused by changes in any single organ, a disease which caused the various organic changes observed." U. S. Ag. Report, 1878, p. 435.

"In every case the colon and cæcum were plainly affected, reddened externally, and internally showing changes, varying from a simple coloration to inflammation and great thickening; in some cases they were studded with petechiæ, while in others there were none; ulcers of various sizes were frequently found, and also thickened, fibrous, concentric patches.

"The cavity of the thorax, in every case, contained a considerable quantity of turbid, bloody liquid. The pleuræ were generally thickened and covered with false membranes; the lungs were constantly found inflamed, occasionally in a few small spots only, but generally the greater part of the lung tissue was involved." U. S. Ag. Report, 1883, p. 437.

"NATURE OF THE DISEASE.

"Is the affection a general or local one? In other words does the disease originate from functional or organic disorder of any particular organ, or apparatus, or are the anatomical lesions developed secondarily, as the consequence of a general infection?

"This question, as regards the disease under consideration, can now be answered in a definite and scientific manner. (! ! !)

"Indeed, when we consider that the first symptoms, and the one preceding all others by several days at least, is an increase in temperature; that when localized a great variety of organs belonging to different systems and apparatus are involved; the lungs, pleuræ, bronchial tubes, heart, liver, stomach, intestines, spleen, kidneys, bladder, and skin; that there are considerable changes in the blood, as shown by imperfect coagulation, solution of the coloring matters, and blood extravasation; there can scarcely remain a shadow of doubt that the trouble is not a local, but a general one." U. S. Agricultural Report, 1878, p. 439.

I wish now to call attention to some evidence quoted from Mr. Salmon's experiments, given in his report of 1885, when he described his "hog cholera" germ of 1886 as the one specific microbe of swine plague. I shall show that he then produced pneumonia as frequently as intestinal lesions, and that he then thought both belonged to the picture of one and the same disease.

He says:

"The second pig (No. 112) inoculated with the same culture (the germ of hog cholera, which does not cause pneumonia, but does cause intestinal lesions in 1886) died in the morning of December 12th.

* * * The spleen was enlarged. * * * On the surface of the lungs numerous dark red spots corresponding to hepatized lobules. These were found throughout the lung tissue.

"The large intestine was generally congested and covered with dark red points. No ulceration." U. S. Ag. Report, 1885, p. 203.

"No ulceration"!

"There was a moderate quantity of watery serum in both pleural sacks. The lungs were normal, with the exception of a small anterior lobe on each side, which was hepatized. Very severe lesions were found in the intestinal tract. This was evidently a very severe case of swine plague." *Ibid.*, p. 195.

Lung disease again!

"On Dec. 5th, two pigs were inoculated (Nos. 121, 140) with a pure liquid culture. No. 121 died on the morning of Dec. 12th. In this case the kidneys and lungs seemed to have suffered the most, if we exclude the lymphatic system." *Ibid.*, p. 205.

"No. 140 died Dec. 18th. The autopsy revealed a very severe case of swine plague. It differed from the preceding case in the presence of extensive ulcers of the large intestine. The lungs were congested and hepatized anteriorly." *Ibid.*, p. 206.

Still hepatization!

"It goes far to prove that the majority of the animals affected with swine plague die from the sequelæ of the disease, and but few from the direct effects of the microbe multiplying in the blood-vessels of the internal organs." U. S. Ag. Report, 1885, p. 211.

What then is swine plague—a pneumonia, or enteritis, or a blood poison, as I have asserted?

Again, Mr. Salmon says of the lesions after death:

"The lungs are in many cases normal, both in cases of rapid death and protracted disease. We have seen many cases in which perfectly sound lungs accompanied extensively ulcerated intestines. In a moderate number of acute, virulent cases the lungs are, in general, collapsed and pale. Under the pleura, however, there are seen small patches of a dark red color, which correspond to limited regions of dark, hepatized tissue, not much more than a fourth of an inch in diameter. These foci are also found throughout the lung tissue in greater or less abundance. In the advanced stages of chronic swine plague the major part of the lungs may be completely hepatized." *Ibid.*, p. 222.

"The fact that it is difficult to demonstrate the presence of the bacterium of swine plague [hog cholera, 1886-7-8] in chronic cases
* * * in which the ulcerations in the intestines are
already far advanced cannot be emphasized too much.

"Chronic swine plague must henceforth be looked upon as an after stage, independent of the disease itself, and caused by intestinal lesions, the indirect result of the growth of the bacterium in the blood-vessels of the mucous and submucous tissues." Report 1885, p. 211.

Compare the above with some of Mr. Salmon's most recent utterances, such as :

"When cultures of this germ, or the spleen and intestines of hogs which have died of hog cholera, are fed to susceptible pigs, there are produced the most remarkable and extensive ulcerations of the intestines.

"Swine plague is a disease in which the principal lesions are found in the lungs ; in hog cholera it is the intestines.

"We have here two diseases, therefore, clearly defined, and produced by entirely different organisms. But they are not always uncomplicated, since we occasionally find both of them in the same herd at the same time." *Journal of Comparative Medicine*, l. c. pp. 140, 143, 144.

To my mind the something else has become much more "complicated" than the swine plague. In 1885, we have "chronic swine plague," caused by "intestinal lesion"; while in 1888, we have "hog cholera," caused by the same bacterium, but the "complicated" peculiarity is, that the disease became acute in 1886, as hog cholera, and it has become acutely painful to understand how this "complicated" condition of things came about.

Perhaps Mr. Salmon can explain ! It is certainly beyond the scope of my ability, notwithstanding I have spent nearly two years in search of the very "complicated" condition of which Mr. Salmon, apparently, knows so much.

These quotations are certainly sufficient to show that in his last report but one Mr. Salmon had no idea that two swine plagues existed in this country. They also show that in his inoculation experiments he produced pneumonia, even in cases where there was no ulceration of the intestines, as well as they show that both sets of lesions can and do occur in one and the same animal.

Now what kind of a micro-organism was the cause of this one disease in 1885?

In his "conclusions," Mr. Salmon tells us that :

"The preceding investigations definitely settle [Alas ! Mr. Salmon again unsettles the whole question in his next report, 1886 !] certain controverted points concerning the etiology of swine plague.

"Swine plague [not hog cholera!] is caused by a specific microbe multiplying in the body of the diseased animal." Ibid., p. 229.

What then was this microbe?

This story is so interesting, although so marvelously contradictory, that we must let Mr. Salmon again tell us about this famous nondescript.

"THE BACTERIUM OF SWINE PLAGUE—1885.

"In at least twenty-five cases of undoubted swine plague, [the reader will please bear in mind that Mr. Salmon says "of undoubted swine plague," and also remember that "these investigations definitely settle" many mooted questions, as well as that there is no mention of any new disease known as hog cholera] bits of spleen tissue * * * were found to contain the same microbe in greater or less abundance. Plate III., Fig. 1. When stained * * * * the greater number present a center paler than the periphery. The darker portion is not localized at the two extremities, as in the bacteria of septicaemia in rabbits, but is of uniform width around the entire circumference of the oval." l. c., p. 212.

I must now request the reader to compare, most critically, the above description with the very latest of the same object, but this time it is not the germ of swine plague, but of that peculiar psychosis of Mr. Salmon's—"hog cholera." The reader will find that "the darker portion" is no more of a "uniform width around the entire circumference of the oval," but that the nonentity is slowly but surely approaching the characteristics of the genuine germ, as I predicted it would about a year ago. Mr. Salmon now tells us "that a dark border extends entirely around the germ. Although it may be thicker at the ends than at the sides, it does not give the distinct appearance of polar staining that is seen in the germs of rabbit septicaemia." In his last report, of 1885, Mr. Salmon was not so (in) "distinct." He was positive "that the darker portion is not localized at the two extremities, as in the bacteria of septicaemia in rabbits, but is of uniform width around the entire circumference of the oval."

On Plate I., Fig. 3, of this report, I have given an exact copy of Mr. Salmon's swine plague nonentity—illustrated Plate III., Fig. 1, in his report of 1885, and on Plate I., Fig. 4, a copy of his illustration, Plate III., Fig. 1, of his report of 1886, in which he pictures his germ of his hog cholera. I especially desire my readers to com-

pare them with the originals, and then ask themselves, honestly! if there are not marked departures in the later from the prior illustration of what Mr. Salmon claims to be one and the same organism? Then, I desire the reader to compare the illustration of this same germ of 1886—the hog cholera psychosis—with that of the real germ of our swine plague, again reproduced from Plate III., Fig. 2, of Mr. Salmon's report of 1886, and he will see how rapidly Mr. Salmon is losing his ocular myopia, though the mental still persists.

However, it is always best to let a man speak for himself, and so we will quote the original remarks of Mr. Salmon:

"The bacterium of hog cholera, as studied in our laboratory, is found chiefly in pairs, appearing as elongated ovals, from 1.2 to 1.5 micromillimeters in width. Occasionally longer elements are observed, reaching 1.8 micromillimeters in length, and even longer under certain conditions. In liquid media the bacterium is motile and its movements resemble very closely bacterium termo. That the bacterium which we have studied is not bacterium termo is shown by the fact that it does not liquefy gelatine and that there is not the slightest putrefactive odor emitted from the culture containing it, and also by its pathogenic effects. This germ may be stained with an aqueous solution of methyl-violet and various other aniline stains. When colored with methyl-violet, particularly when in cover-glass preparations made from the organs of affected animals, the central portion is much paler than the periphery. A dark border extends entirely around the germ, and although it may be *thicker at the ends than at the sides*, it does not give that distinct appearance of polar staining that is seen in the germs of rabbit septicæmia." *American Vet. Review*, l. c., p. 8., April.

Let us now turn to some of Mr. Salmon's points of differentiation between his germ of hog cholera, and that of swine plague proper. It will be found to excel "*Puck*" in ridiculous inconsistency.

SOME EXAMPLES OF MR. SALMON'S INCONSISTENCIES AND CONTRADICTIONS WITH REFERENCE TO HIS GERMS OF "HOG CHOLERA" AND THE SWINE PLAGUE ORGANISM.

First I will show that Mr. Salmon gives two entirely different descriptions of his "microbe" of hog cholera.

1. "When stained with an aqueous solution of methyl-violet the greater number present a center paler than the periphery. The darker portion is not localized at the two extremities, as in the bacteria of septicæmia in rabbits, but is of uniform width around the entire circumference of the oval." Report 1885, p. 212. See Plate I, Fig. 3.

2. "The bacterium of hog cholera, as studied in our laboratory, may be stained with an aqueous solution of methyl-violet and various other aniline stains. When covered with methyl-violet, particularly when in cover-glass preparations made from the organs of affected animals, the central portion is much paler than the periphery. A dark border extends entirely around the germ, and although it may be thicker at the ends than at the sides it does not give that distinct appearance of polar staining that is seen in the germs of rabbit septicæmia." *Am. Vety. Review*, 1. c., p. 7. See Plate I., Fig. 4.

3. "The appearance after staining is almost equally marked, providing we examine stained cover-glass preparations, made from the organs of affected animals. In the one case (hog cholera) we have a long, oval element, with a dark border and pale center; in the other (swine plague) we have a short, oval element, staining darkly at the extremities and with an unstained transverse band across the median portion." 1. c., p. 12.

The reader will please notice that in the first description we have no staining of the body of the germ, that part being entirely clear, as Mr. Salmon has illustrated it. In the second, "the darker border" may be "thicker at the ends," though not so "distinct as in the germs of rabbit septicæmia." While in the third case Mr. Salmon again goes back to his original description of 1885, and even contradicts himself in the article quoted, where he admits this object does stain some at the ends.

If these quotations are not the most direct evidence of mental "jugglery," it would be hard to find such. Here we have, directly before us, two different descriptions of one and the same organism, so different, in fact, that no one could recognize them as relating to the other. It is also a singular evidence of Mr. Salmon's appreciation of his audience at Baltimore, that these contradictory descriptions should have been given in his address to them, and again published for their subsequent edification. It is also remarkably creditable to

the ability of the editors of the *American Veterinary Review* and the *Journal of Comparative Medicine*, in which this address was also published, that neither of them should notice such absurd contradictions and call editorial attention to them.

These editors should be praised for their critical powers, for they have never seen that even in his report of 1886 such contradictory assertions as the following may be found. It must be remembered that Mr. Salmon, in other places, is positive that the real swine plague germ is the specific cause of pneumonia only. Yet he says:

"The evidence which has been brought forward in preceding pages, that the microbe described is the cause of pneumonia in pigs, which is, therefore, from an etiological stand-point, wholly different from hog cholera, is not yet conclusive, and will require further investigation." (!!) 1886, l. c., p. 682.

Of this same pneumonia he says, in another place: "It seems that this hitherto unknown disease of swine," (p. 677) but entirely forgets that on page 618 of the same report he had already written, "in view of the fact that another bacterium has been recently discovered," etc., and that he also admitted the fact of that discovery in November, 1886. What then can we say of Mr. Salmon's honesty? That this assertion about this "unknown disease" (1886) is an absolute falsehood will be seen by reference to quotations from Mr. Salmon's publications. It is also manifested by the following from Dr. Detmers, whom Mr. Salmon utterly ignores:

"Although the morbid processes of swine plague can have their seat in almost any organ or part of the body, it must be considered as characteristic of the disease that the lungs invariably are more or less affected, and constitute, in a large number of cases, the principal seat of the morbid processes.

"At any rate, in over two hundred *post-mortem* examinations, I found, in every case, more or less of that peculiar hepatization characterized by its distinct limits, by its different appearance and color, according to its age, in different parts of the lungs, and sometimes even in adjoining lobules, and by the small red or red-brown specks of extravasated blood, usually exceedingly numerous in those parts of the lungs not yet fully hepatized, or in the first stages of hepatization.

"So, I have come to the conclusion that hepatization of at least some portion of the lung tissue must be considered as a never absent morbid change, characteristic of the disease, and that no swine plague

is existing where the lungs are not morbidly affected, or when they are found to be in a normal condition.

"If other parts were not also frequently affected, and in some cases even more than the lungs, swine plague might be called a bacteritic pneumonia." Report 1880-81, p. 155.

HOW THE "HOG CHOLERA MICROBE" DIFFERS FROM THAT OF SWINE PLAGUE IN MANNER OF ACTION, ACCORDING TO MR. SALMON.

Hog Cholera.

"This germ is fatal when inoculated in mice, rabbits, guinea-pigs, and pigs, and occasionally so when given to fowls and pigeons in large doses.—*Am. Vet. Review*, l. c.

"Taking the germs first, we find that of hog cholera to be motile, while that of swine plague never presents any evidence of motility. This difference is a very striking and radical one.—l. c.

"The hog cholera germ grows

Swine Plague.

"That it was not the bacterium of hog cholera was shown by an utter want of pathogenic properties when inoculated into mice and rabbits." U. S. Ag. Report, 1886, p. 662.

"Rabbits, mice, and pigeons were thus shown susceptible when inoculated with the quantities above mentioned." Ibid., p. 665.

"This microbe was, therefore, fatal to mice, rabbits, and pigs." Ibid., p. 662.

"Mice are killed by the hog cholera germ in from eight to sixteen days, by the swine plague in from two to six days.

"Rabbits die from hog cholera inoculations in from six to nine days, but they only live from twenty hours to six days after inoculation with swine plague."—*Am. Vet. Review*, l. c.

"One grew in both tubes, which was more carefully examined, because it resembled the bacterium of hog cholera very closely. In liquid media it is actively motile, and simulates the form of a bacillus." 1886, p. 661.

"In one of the tubes the motile bacterium just described." Ibid., p. 662.

"On potatoes, a thick, straw-

actively on the cut surface of potatoes, while the other microbe refuses to grow at all in that condition."—*l. c.*

colored, shining layer of nearly smooth surface forms, which grows very vigorously and gradually covers the entire cut surface of the potato with a layer 2^{mm} thick. This growth is brighter in color and more abundant than appears in the potato culture of the bacterium of hog cholera." *Ibid.*, p. 661.

"The hog cholera germ inoculated directly into the lung tissue produces no hepatization or marked lesion of any kind, but is followed by the development of a hemorrhagic or ulcerative case of hog cholera.

"In this outbreak, then, more than half of the animals had some defect of the lungs.

"We have found, however, that in the lung lesions accompanying hog cholera." *Ibid.*

"The germ of hog cholera produces fatal effects when inoculated into mice, rabbits, guinea-pigs, pigeons, and pigs." *Ibid.*

"We soon found that there was no indication for attenuating the virus for this purpose, because the strongest virus might be introduced hypodermically with impunity in considerable doses."

Will Mr. Salmon please explain how "fatal effects" could be produced in "pigs" by a "virus" which he "soon found no indication for attenuating"?

Reader, after the careful consideration of the foregoing, what is your opinion? After having previously given you conclusive proofs that this same "hog cholera microbe" of Mr. Salmon's not only was his swine plague germ of 1885, and then produced the pulmonary lesions as well as intestinal, have I not again shown you that the same object, according to Salmon, produces the same lesions when it is made the germ of hog cholera alone?

Has Mr. Salmon given a particle of evidence showing that this germ produces a single lesion, or does a single thing that that of swine plague does not?

Then, reader, what is it?

I will answer for you: An imposition!

Not content with giving such condemnatory evidence against himself, Mr. Salmon has even gone so far as to pile it up so high that his eventual crushing into a worse specimen of nonentity than his "hog cholera" psychosis is but a question of time. He has done this by sending a lot of cultures of his "nonentity" to European investigators, all of whom say that they have never met with it, and that it does not occur in any case of swine plague known to them in their respective countries. They certainly "settle the question" that there is no such pathogenetic germ known to them, and I think the question has also been sufficiently settled that the real swine plague, with its one ovoid, belted germ, is either known to them all, or that there is evidence that it exists in their respective countries, except the, at present, somewhat peculiar character of the testimony from Sweden and Denmark with regard to the micro-organism, of which I have no fears that the little inconsistencies will eventually be cleared up.

TESTIMONY OF EUROPEAN INVESTIGATORS OF THE NON-EXISTENCE OF SALMON'S "HOG CHOLERA MICROBE" IN SWINE PLAGUE.

"Having referred to the main points of this destructive malady which have been elucidated by the experiments made under my direction," Mr. Salmon says, "I shall conclude with a few opinions of European investigators in reference to points which have been contested.

"You will remember that it has been asserted in the most emphatic terms that there is but one disease in this country which goes under the name of cholera, and that it is identical with the Schweineseuche of Schütz. To settle this question I have sent to the leading investigators of Europe cultures of the germ of hog cholera, together with copies of my reports and asked for their opinions. I trust you will bear with me while I read what they have since written on this subject:

"M. Nocard, after receiving this culture, says editorially in the *Recueil de Médecine Vétérinaire* for Jan. 15, 1888, p. 8:

"Recapitulating, we see that under the name of Rouget, mal rouge,

erysipelas, etc., etc., there may be confounded at least three very different maladies:

“First. The rouget, properly speaking. Second. The hog cholera of the Americans, probably identical with the swine fever or infectious pneumo-enteritis of the English, and without doubt also with the diphtheritis of the pig recently observed in Sweden and in Denmark. Third. The infectious pneumonia, or Schweineseuche of the Germans, or swine plague of the Americans.’ [It will be noticed that Nocord makes no mention of micro-organisms. On the contrary, every word he says is in favor of but one disease, and contradicts Mr. Salmon. He is simply quoting from books, however, and has no value as an authority, although his conclusions are correct.—B.]

“Dr. Rietsch, of Marseilles, who has recently studied the outbreak of swine disease near that place, writes under date of Feb’y 15:

“‘The microbe which I send you is not the same as that you discovered in hog cholera. The hog bacterium grows at 20° more slowly than mine and at a lower temperature there is reached a point where the hog microbe no longer grows, while our bacillus still forms very fine colonies.

“‘The gelatine colonies differ in appearance.

“‘Finally your hog microbe is more motile and a little smaller, it seems to me, in the same condition.

“‘The microbe of Marseilles differs from that of Schütz by its motility, that of Loeffler-Schütz being non-motile. The disease of Marseilles seems also to differ from the Schweineseuche by the absence of œdema at the point of inoculation, by slower progress of the disease, by the pathological phenomena being found in the intestine almost always, while Schütz observed almost nothing in the intestine and never mentioned ulceration.’

“In a subsequent paragraph he adds, that my hog cholera microbe certainly differs much more from those of septicaemia of rabbits, Schweineseuche of Loeffler-Schütz, Wild-senche of Kitt, and fowl cholera of Perroneito and Pasteur than these germs differ from each other.

“The germ discovered by M. Rietsch at Marseilles is very closely allied to our hog cholera microbe and probably differs no more from it than specimens of our germ differ when obtained from different outbreaks in this country.”

[1st. We see that Rietsch claims that the swine plague germ discovered by him has “motility,” which corresponds with my own opinion. The cause of this difference of opinion about “motility” has been discussed in my treatment of the germ of swine plague.

2d. M. Rietsch seems to be unaware of the fact that the Loeffler-Schütz “Schweineseuche” is not a new disease, but the well-known

"Wild-scuche;" but he is the first European observer to call attention to the fact that the "œdema" which characterizes it does not occur in the true swine plague, which is the disease he studied at Marseilles. He, also, has not seen that Schütz's second series of hogs—those from Pülitz—were probably diseased with the swine plague and not the Wild-scuche; hence, the same disease as has been studied in this country, Great Britain, and France.

3d. Rietsch's germ is undoubtedly the same as that of Detmers, Cornil, and myself, for he tells us "that in later cases pneumonia was frequently present," while in the first observed the intestines suffered most.—B.]

"Dr. Cornil, who is now a member of the French Senate, and who certainly is one of the very highest authorities on micro-organisms in France, writes me, under date of Feb. 10, 1888, as follows:

"We have had occasion to study, M. Chantemesse and I, the disease of swine that you described two years ago under the name of swine plague and in your last volume under the name of hog cholera. —"et par Salmon successivement sans les noms de swine plague et de hog cholera."* We have observed an epidemic in June last at Paris, and at the end of the year another epidemic at Marseilles. We have verified the accuracy of your description and we are about making experiments of vaccination.'"

Cornil's remarks have relation to swine plague as described by Salmon in 1885, and not to Mr. Salmon's hog cholera, or his germ of that disease. He seems to know nothing of Detmers' and my work, nor does he realize that Mr. Salmon has separated that disease distinctly from hog cholera. It will be remembered that Cornil produced the combined pulmonary and intestinal lesions of swine plague by injecting pure culture of the same germ described by Detmers and myself into the lungs of a pig, and that he found some germ in the tissues of that same pig subsequently. It will be particularly noticed that Cornil does not say one word about Mr. Salmon's hog cholera, new microbe.

And now comes the "clinger" with which Mr. Salmon fondly hoped to annihilate all contradiction. It is as follows:

"I have one other letter to which I attach the very greatest importance, because it comes from the highest authority in the world on pathogenic bacteria. Under date of Feb. 23, 1888, Dr. Robert Koch writes:

* *Comptes Rendus*, Tome CVI., p. 612., Feb., 1888.

“‘The culture of hog cholera sent to me came in good condition, and I directed Dr. Esmarch, one of the assistants in the Hygienic Institute, to make a few experiments with it. He was able to confirm all of the results obtained by you in inoculating and feeding mice and guinea-pigs. This micro-organism does not correspond with any of the species of pathogenic bacteria known here, particularly not with those found in swine diseases.’”—*American Veterinary Review*, l.c., pp. 18, 19.

What does Koch's evidence show?

Nothing, but that some unknown germ was sent him that had some kind of effect on “mice and guinea-pigs,” but not that it had any on hogs. It does show that it does not occur in any swine disease known to him as occurring in Europe, and all the evidence quoted by me goes to show that the so-called swine plagues there are one and the same with that in this country, except the Wild-seuche and Rouget.

Before closing these remarks I desire to call attention to a very singular omission of Mr. Salmon's with regard to what he claimed was a very essential differentiating point in the pathogenic action of the true swine plague germ from his hog cholera nonentity, in the article in the *American Veterinary Review*. In his report of 1886 he says that the swine plague germ causes “cirrhosis of the liver.” In his latest publication he knows nothing about that. Was it because I had shown the utter absurdity of such a thing in an acute infectious disease like the swine plague?

I have lately received a letter from Prof. William Welch, pathologist of Johns Hopkins University, at Baltimore, Md., which is of essential value to the point in dispute. From it the reader will see that, though working so near Mr. Salmon's center of operations, Dr. Welch's discovery confirms my own, and that of Dr. Detmers, though he follows Locffler in saying the organism does not grow on potatoes, but on the contrary we have the authority of Cornil, Rietsch, Hueppe, and Kitt, as well as my invariable success with freshly procured germs, that both the organisms of the swine plague and the Wild-seuche do grow on potatoes. Very old agar-agar cultures will still live on potatoes, but generally have lost their power of rapid proliferation on potatoes, and do not then give the same peculiar greyish color.

Prof. Welch's communication is as follows:

“JOHNS HOPKINS UNIVERSITY,
BALTIMORE, MD., April 18, 1888.

“MY DEAR SIR—I have made an investigation of the organism found in a local epidemic of swine plague in this city. This organism seems to correspond essentially with that described by Loeffler.

“Yours truly,

“W^M. H. WELCH.”

It is due to justice to say that Prof. Welch does not yet admit that his germ is the same as mine, but I have no doubts on that point, potatoes or no potatoes.

The interesting question now is, What kind of an organism is Mr. Salmon sending round as the “specific microbe of hog cholera”?

It shows a singular want of scientific exactness that Rietsch, of Marseilles, who had it while making his own investigations, does not give any description of this thing. It has been absolutely impossible for me to get any culture of this organism, but last summer Dr. Detmers sent me a slide marked as follows: “Bacterium Salmonidi,” on one label, and on the other, “Doctor B. Persh, culture in gelatine. 10, 24, 86.” This organism bears no relation to anything described by Mr. Salmon, either as the germ of swine plague in '85, or of his hog cholera in '86, except, perhaps, in outline morphology; it is a small ovoid organism, about three times as long as wide, and has rounded ends, but colors, or is colored, entirely, and it does not seem as if the tinction had been too strongly applied. Sometimes, when in process of fission, there does seem to be a very slight clear space in the middle of the body, but in no way does it resemble Mr. Salmon's description or plates, with the exception of the very latest, where the ends do color somewhat.

Is this Mr. Salmon's “hog cholera” bacterium?

As to that I know not, but I will permit Dr. Detmers to tell his own story about it.

“DR. SALMON'S SWINE PLAGUE BACTERIA OF 1885.

“Sometime in October, Dr. Persh, of Philadelphia, had the kindness to send me two tubes containing cultures of Dr. Salmon's swine plague bacterium, one of them in nutrient gelatine and the other in agar agar. With the gelatine culture, which, on microscopic examination, proved to be perfectly pure—that is, a culture containing but one microbe, as can be proved by a photograph taken from a vesuvium-stained slide made from that culture—I inoculated, at 10 A.M. of October 24, a

young white rabbit, nearly full grown, and perfectly healthy. It belonged to the same litter as No. 2 and No. 3, and will be called Rabbit No. 1. (It ought to be superfluous to state that all the animals experimented with were separated before they were inoculated, and have been kept in cages that had no connection or communication whatever with each other.)

“October 27, 5 P.M.—Rabbit No. 1 appears to be sick.

“October 28, 8 A.M.—Rabbit No. 1 is decidedly sick; arches its back, is very quiet, permits to be touched, has no appetite, but does not droop its ears. No local reaction; that is, no swelling, congestion, or inflammation whatever can be discovered in the ear, in which the inoculation has been made.

“October 29—Rabbit No. 1 is drowsy, in a soporous condition, and dies at 6 P.M., without a struggle.

“October 30, 8 A.M.—*Post-mortem* examination in presence of Prof. R. W. Lazenby, who took the following notes while I made the following *post-mortem* examination: Rigor mortis fully developed. After skin is removed, no discoloration of muscles and subcutaneous tissues. Condition as to flesh yet good, and considerable fat of a normal color. On opening the chest a small patch of the anterior right lobe of the lungs, a patch hardly as large as a silver three-cent piece, brownish colored, and containing some exudation. Blood in veins and in heart very dark, and not coagulated. All other parts of lungs, and whole left lung, perfectly normal, though not fully collapsed. Liver about twice its normal size; so the kidneys. Spleen about $2\frac{1}{2}$ or 3 times its normal size—over 3 inches long and very thick—and of a purplish-black color. Liver tissue very soft, breaks on touching (slightly pressing) it with the finger. Contents of gall-bladder dark colored; lymphatic glands nowhere enlarged or morbidly changed. Nothing abnormal in the intestines, except that the mucous membrane of the colon is rather dark colored, but as this dark color is diffuse, and not at all in patches, and as the animal has been dead fourteen hours, it must be looked upon as a *post-mortem* change.

“The morbid changes, therefore, were confined to the spleen, the liver, and the kidneys, and a very small portion of one of the anterior lobes of the lungs.

“At the *post-mortem* examination blood was taken from the heart by means of vacuum tubes, in the manner already described, and the spleen, a portion of the liver, and one kidney were put in diluted alcohol, and a week or two later half of the spleen, half of the kidney, and a piece of the liver sent Dr. J. E. Reeves, of Wheeling, W. Va., for mounting. The large and very thin sections of all three tissues, beautifully double stained and mounted in Dr. Reeves' superior manner, show on every slide numerous bacteria identical to those with which the animal was inoculated, and which were found in the blood

and in the cultures started with the latter, as can be proved by the photographs that have been taken.

"Now the question arises, did rabbit No. 1 die of swine plague? If it did, I must say I have seen many cases of swine plague, but I have never seen a case of that disease that presented features (symptoms and morbid changes) identical, or even similar to those exhibited by the disease of which rabbit No. 1 died. In the first place, the symptoms did not resemble those of swine plague; the period of incubation was rather short, and the blood was very dark, almost black, and not coagulated, notwithstanding that the lungs, with the exception of a very small portion, were perfectly normal, while, according to my experience, the blood, in genuine cases of swine plague, always coagulates, at any rate, to a loose clot, and is never very dark unless the lungs are seriously affected. At least I have always so found it in all cases of swine plague that have come under my observation, and their number is not a small one. Further, the lymphatic glands, always more or less affected in every case of swine plague that I have ever seen, did not show a trace of morbid changes, while the organs found diseased—the spleen, the liver, and the kidneys—are comparatively seldom affected in swine plague, and then in an entirely different way. The disease, most assuredly, was bacteriæmia, and caused by the inoculation; but I cannot identify it with swine plague. If it is identical with it, then I must say, the disease I heretofore pronounced swine plague, or, as the farmers call it, hog cholera, must be bogus; that is, something else. I am compelled to pronounce the disease of which the rabbit died a fatal septic disease entirely different from swine plague." Fifth Annual Report of the Ohio Agr. Expt. Station for 1886, pp. 289, 290.

Now here we have evidence of three facts:

1st. This organism does not correspond to any of Mr. Salmon's descriptions, and only in a slight degree to the latest.

2d. The cultures were pure.

3d. It had septic action on rabbits.

These three facts lead to two questions of great interest:

1st. Was this material from Dr. Persh genuine, and did it come, as said, direct from Dr. Salmon?

2d. Swine plague itself being a septicæmia, may it not be possible that Mr. Salmon has accidentally dropped upon some kind of a septic organism capable of producing the same lesions?

This question has been frequently in my mind of late, for if not so, I do not know how to answer for all Mr. Salmon's wonderfully positive experiments. If not so, then these experiments were never

made, and the whole thing is a concocted farce, or else he has been secretly using the real germ and showing people this other thing. I am determined to force this question to such an issue that the real facts must come out sooner or later, for I, as repeatedly said, have no fears but what my own work will stand every test.

The burden of proof to the contrary now lies with the Agricultural Department of the United States government, or even with congress itself. For my own part, I demand that this test as to the true germ of swine plague, or as to whether there are two porcine pests in this country, as Mr. Salmon has vainly endeavored to show the world, be settled by a competent commission of experts, but not by any one connected with the government in any way. The position I have put the Agricultural Department in should force congress to employ investigators of honorable reputation and some scientific credence in the world, such as Welch, of Baltimore, and Shakespeare and Osler, of Philadelphia, and then this question will indeed be settled and "the evidence furnished be such as will (positively) settle a scientific question of this kind."

The porcine interests of America demand this of congress, and it is to be hoped that the Live Stock Growers' Associations, at their meetings next fall, will take steps that will be more successful in making their demands and rights felt in Washington than they have been enabled to do heretofore. So long as this uncertainty goes on, so long as these apparent contradictions on a simple and easy to be decided matter exist, so long will swine breeders be doubtful of the best methods of preventing their losses, no matter from what source they emanate. Still, I am in hopes that the evidence here furnished is of such a scientific as well as practical kind that when I come to treat upon prevention the swine breeders will see truth enough behind the mists of incompetency and misrepresentation, which I have endeavored to sweep away, to give them confidence in my statements and follow out advice and methods which I assure them will finally lead to reducing their losses from swine plague to a bearable minimum.

EVIDENCE THAT THERE IS BUT ONE SWINE PLAGUE IN THE UNITED STATES FROM OTHER OBSERVERS.

I have previously given the testimony of Klein and Walley, which is equally positive and conclusive against Mr. Salmon's peculiar psychosis.

We will now quote some more of a similar character.

Dr. D. W. Volles, 1879, of Indiana, says:

"Memoranda of thirty dissections made from fifteen separate and distinct herds fairly representing the disease.

"In every case, without exception, disease of the lungs was present, varying in degree from slight congestion to complete softening from suppurative inflammation.

"In eight cases adhesions between costal pleuræ and the lungs.

"In four cases small patches of ulceration of the mucous lining of large intestine!

"In all cases the liver presented a darker hue than natural.

"In four cases there was slight congestion of the kidneys. In one case evidence of fatty degeneration, in all others the organ indicated a healthy condition." *Veterinary Journal*, Vol. IX., 1879, p. 186. U. S. Ag. Report, 1883, p. 424.

Dr. Wm. Osler, late of McGill College, Montreal, but now connected with the Medical Department of the University of Pennsylvania, made some study of swine plague in an outbreak that occurred in the former place! His necroscopical observations were made upon nineteen animals. From other remarks I select the following, which are all that are necessary:

"Successful experiments with the caseous matter from the bronchial tubes demonstrates, for the first time, that the contagion is also contained in the lungs.

"Two facts are very remarkable about the condition of the intestinal lesions:

"I. The absence of ulceration in most of these cases.

"II. The very slight hyperæmia, or injection of the mucous membrane, about the plaques." *Veterinary Journal*, Vol. VI., p. 399.

Under the heading "Pleuro-pneumonia in Pigs," is to be found an article in the *Veterinary Journal*, Vol. 2, p. 350, by a Mr. A. R. Colman, from which I quote the following extracts, in order to show that the disease was undoubtedly swine plague and not "pleuro-pneumonia."

A Mr. S. bought two hundred green hogs in the United States for fattening in Canada. They did not appear well soon after their arrival, and the author says:

"They took readily enough to the whey (from the cheese factory) and all appeared to be doing well, and continued to do so for a few weeks, with the exception that some were troubled with a cough. But in a very short time afterwards worse symptoms appeared; they got off their feed; diarrhoea set in, or, in some instances, very obstinate constipation, with difficult breathing and vomiting; they soon commenced to die off rapidly, so that the attendant's duties of feeding were somewhat perverted, and grave-digging and burying claimed the greater share of his attention.

"The following symptoms were observed: When disturbed, a very distressing, dry, hacking cough came on; some would vomit; rigors and dullness were more or less present; there was great general debility; most of them offered a very emaciated and tucked-up appearance about the abdomen. Some of them were constipated, others had diarrhoea, and some exhibited a red or purplish appearance under the abdomen, particularly between the fore legs and under the neck.

"There being no dead hogs above ground, and as evening was approaching, we did not make a *post-mortem*, but from general appearances and the history were led to think that the disease was what is generally known as "hog cholera."

"The next day we went again and opened two or three of the dead hogs, and killed one of the affected ones for examination.

"In all we found the greater portion of the lungs more or less congested, much consolidation in some, involving both the lung tissue and pleura. In most there was a considerable quantity of pus in the lungs; and in some were abscesses ulcerating completely through the lung substance. There was likewise a considerable quantity of effusion present, and the lungs were also more or less attached to the side.

"All the other organs were normal. (!!!?)

"One hundred and twenty died out of a herd of two hundred."

The writer of the above was then a student at the Ontario Veterinary College.

Prof. James Law, of Cornell University, of Ithaca, N. Y., says:

"The lymphatic glands of the mesentery and the abdomen generally may be said to have been uniformly altered. Those in the vicinity of the congested or ulcerated patches of the intestine were usually of a dark color, confined to the surface of the gland, or, in the worst cases, extending through its entire substance. * *

"Though the lungs never entirely escaped, in one case only was the entire lung hepatized. Exudation and consolidation of the lung tis-

sue were in a few cases confined to the anterior lobes, but as a rule the posterior lobules only were affected. In some cases exudation was confined to the interlobular spaces, which accordingly appeared as broad lines, circumscribing the lighter colored lobes." U. S. Ag. Report, 1878, pp. 369-70.

It should be remarked that Prof. Law had not then studied the disease as it occurs in the field to any extent, but that his observations were largely made upon hogs inoculated with material sent to him, still we see that his evidence goes to show but one disease.

To the foregoing testimony it may be objected, by some quibblers, that no bacteriological investigations were made, hence I now desire to support it by quotations from one who has studied swine plague in this country over a larger extent of territory than any other person, and whose claims as the first discoverer of the germ of swine plague I have endeavored to show, in the spirit of simple justice to a man whom I respect and admire for the wonderful work he has done under most adverse circumstances. I will say that I have lately visited Dr. Detmers, and assured myself of the correctness of his bacteriological work by actual inspection. Though he tells his own story at the conclusion of this chapter, I will begin his evidence by quoting from a letter dated May 4, 1888, in which he says:

"We agree perfectly on the essential points. I have made between three and four hundred autopsies in seven different states, and found but one disease and one germ. The second exists nowhere but in Mr. Salmon's cranium."

In 1878 Dr. Detmers wrote:

"That the morbid processes, although in all cases essentially the same, are not restricted to any single part or organ, or to a set of organs, but can have their seat almost anywhere; in the tissues of the lungs, the pleuræ, the heart, the lymphatic system, the peritoneum, the mucosæ, especially that of the intestines; in the liver, the spleen, and even in the skin. Only the pulmonic tissue and lymphatic glands are invariably affected." Report U. S. Dept. Agriculture, p. 323.

The last assertion is a little too positive.

In 1883 he again said:

"The morbid processes, although everywhere essentially the same, can have their seat in many different organs or parts of the body, and produce, therefore, a great variety of morbid changes. The disease in

consequence very often presents a somewhat different aspect in different animals. The constant morbid changes consist—

“1. In a more or less perfect hepatization of a larger or smaller portion of the lungs, or a more or less extensive accumulation of blood, blood serum, and exudation in the pulmonal tissue. In some cases, especially those in which the morbid changes were of recent origin, no fully developed hepatization had yet been effected, the lungs were merely gorged with exudation, the texture not yet being destroyed or seriously changed, but innumerable small red spots, indicating incipient embolism, were visible to the naked eye. In other cases hepatization was present only in certain insulated places, while in others it complicated whole portions of the lungs. In all cases where the hepatization was very limited it was found in the anterior lobes, principally. In some animals, old or, so-called, grey, more recent, or brown, and very new, or red, hepatizations were frequently found side by side, or in more or less distinctly limited patches, showing distinctly that the morbid changes had not been produced at once, but at several intervals.

“2. The lymphatic or mesenteric glands were invariably found more or less enlarged. In some cases they presented a brownish, or even blackish color, and contained effusions of blood in sufficient quantities to push aside the normal glandular tissue.

“3. The trachea and bronchial tubes contained, in all cases, more or less frothy mucus, the membrane being hyperæmic and more or less swollen.

“4. The pulmonal and costal pleuræ, the mediastinum and pericardium, presented morbid changes in nearly every instance. In some animals these membranes appeared to be smooth, but either the thoracic cavity or the pericardial sack contained a smaller or larger quantity of a straw-colored fluid. In a great many cases these membranes were coated, to a varying degree, with plastic exudation. In some a more or less firm adhesion had been effected between the costal and visceral pleuræ, or between the latter and the diaphragm, while in a few the whole extent of the lungs was attached to the inner surface of the thoracic cavity. In one case the external surface of the heart was firmly attached to the pericardium.

“5. In nearly every animal the heart has been found more or less affected in one way or another.

“6. In forty-eight out of fifty-three cases, morbid changes have been found in the cæcum and colon. The same consist in peculiar growths or ulcerous tumors on the mucous membrane of the intestine. They are of various sizes, nearly round or somewhat irregular in shape, more or less elevated above the surface of the mucosa, and frequently, especially the larger and older ones, dark pigmented on their surface. They vary in size from a pin's head to a quarter of a

dollar. The smaller ones are usually of an ochre color, and project but slightly above the surface, while the larger ones are of a greyish-black-brown color, or even black, and extend considerably above the surface, and have usually a slight cavity in the center. Their surface is made up of a granular detritus, while their stroma consists mainly of dense connective tissue.

"7. Morbid changes in the serous membranes of the abdominal cavity. In some cases the peritoneum and serous covering of the intestines was perfectly smooth, but a varying quantity of straw-colored serum was found in the abdominal cavity. In others, adhesions between the intestines and the walls of the abdomen, or between the folds of the former, were present.

"8. Slight changes (congestion) were found in a few cases in the kidneys. (!!?)

"9. In a great many cases, red or purple spots, or patches, and even continuous or confluent redness of a purple hue presented itself in the skin on the lower surface of the body, between the legs and behind the ears. In some cases whole pieces of skin had become degenerated, and sloughed off, leaving an ulcerated, exposed surface." U. S. Ag. Report, pp. 337, 442.

The following letter from Dr. Detmers will form fitting testimony to this side of the argument:

"*Dr. F. S. Billings, Director of the Patho-Biological Laboratory, University of Nebraska:*

"DEAR SIR—In answer to your favor of May 2d, allow me to say: The swine plague germ of which I recently sent you a photograph (Plate III., Fig. 2.) and some time since a slide, is the very identical organism which, in September, 1878, I found to be the cause, and the sole cause, of that disease of hogs, which I named 'swine plague,' and which the farmers call 'hog cholera.' In my first report to the commissioner of agriculture (Commissioner's Annual Report for 1878) I named it, very appropriately, I think, *Bacillus suis*. In my second report (Commissioner's Annual Report of 1879, page 413), I dropped the name 'Bacillus,' because I was misled by the statements of Cohn and others in their classification of Schizomycetes, according to which 'Bacilli' never form 'Rasen' or zoöglæa masses. As my swine plague germs did form zoöglæa masses, they could, in deference to so great an authority as Cohn, not very well be classed as bacilli. Hence, I preferred to give them no special name whatever. When, however, it became evident that some bacilli do form zoöglæa masses, and that Cohn had made a mistake in the statement above referred to, I did not see any reason why that name should not be taken up again. At any rate, if *bacillus tubercu-*

losis, bacillus anthracis, and Dr. Koch's comma bacillus are bacilli, then this swine plague germ is most assuredly entitled to the same appellation. Still, as long as the nomenclature of bacteria or microbes remains as indefinite as it is, and has been till now, the name is not very material.

"When I made my discovery in 1878 I hinted that swine plague must be a bacteritic disease, but Dr. Schweizer, who assisted me as microscopist failed to find the bacteria—I had to work with an inferior microscope of my own, an instrument not made for bacteriological research; staining processes were not known, and methods of sterilization and of making artificial cultures were yet in a very crude state; besides that, I had no laboratory and was compelled to use the most primitive appliances, because the department of agriculture never furnished me anything, except four years later, two objectives. It is therefore nothing I need be ashamed of now that my descriptions and crude drawings of the swine plague germ were not as accurate and precise as they would be to-day, also that the most approved methods, now known to me, were not employed in my early researches. For two reasons I will not attempt to give a description now. 1. *Because your 'swine plague,' or what is the same, 'hog cholera,' germ and mine are identical, as has been proved by an exchange of slides, and by the photographs I send you, which were taken from a pure culture (No. 17a) in nutrient gelatine; and 2. Because a detailed description would occupy considerable space, and would be perfectly superfluous, because you undoubtedly will say all that is necessary on that topic in your forthcoming report. Where there is no difference of opinion, repetition is useless.*

"If one will peruse my first and second report to the commissioner of agriculture, as published in his annual reports for 1878 and '79, respectively—published, therefore, before Dr. Salmon ever mentioned bacteria, bacilli, or micrococci in connection with swine plague, hog cholera, or any disease of hogs, and will do it without bias and prejudice, he will find that I proved five things: 1. That swine plague, or what is the same, hog cholera, is a bacteritic disease *sui generis*. 2. That it is caused by a specific germ. 3. That this specific germ, which I called bacillus suis in my first report, is invariably present in the blood, urine, mucus, fluid exudations, excrements, and in all the affected tissues of the diseased animals. 4. That the disease is communicated in a mediate way, by infection, and not by mere contact; hence, is *infectious* and not *contagious* in a proper sense of the word. 5. That all the various forms in which swine plague or hog cholera may present itself, belong to one and the same disease—in other words, that *the morbid process, in all cases, is essentially the same.* (cf. Annual Report of Commissioner of Agric. for 1878, page 337, and subsequent reports.)

"When Dr. D. E. Salmon, of the Bureau of Animal Industry, sets up the claim that the disease is contagious; when he asserts that more than one specific germ constitutes the cause of the morbid process; and when he distinguishes two different diseases, which he calls 'hog cholera' and 'swine plague' respectively, and ascribes to each a specific germ, and nearly every year a new one, as the specific cause, he is very much mistaken, and plainly shows that he is shooting at a mark and aiming to hit the bull's eye without seeing the target. When it is so evident, as in the case in question (cf. my reports for '78, '79, 80), that all the various morbid changes met with in this disease, denominated swine plague and also hog cholera, are the result of one and the same morbid process, and consequently belong to the same disease and have the same cause, such a mistake is inexcusable, and can be accounted for only by the supposition that the person who made the same, in the first place, is not practically acquainted with the disease in question, and lacks sufficient knowledge of pathological and physiological processes; and secondly, has endeavored to bring his statements into accord with misconstrued or imperfectly understood (or undigested) statements in foreign literature.

"I can assure you, there is so far but one swine plague in this country; at any rate, in about 400 *post-mortem* examinations made in seven different states of the Union (Ohio, Indiana, Illinois, Iowa, Missouri, Kansas, and Texas), I have invariably found but one and the same disease. The morbid changes ascribed by Dr. Salmon to his swine plague I never found entirely absent, but in some cases more conspicuous than in others, while those he ascribes to (his) hog cholera I found united with them in from twenty-five per cent to seventy-five per cent of all cases, according to season, surroundings, severity of attack, principal seat of morbid process, and, probably, means of infection (cf. my reports for 1878, 79, and 80).

"I frequently found some other morbid changes, changes which, I think, have not at all been mentioned and classified by Dr. Salmon, for instance, sloughing of the skin at various parts of the body, destruction of the nose, of the gums, an affection of the eyes, of the spermatic cords, etc. Do these morbid changes, according to Salmon, belong to hog cholera or to swine plague? That in a disease like swine plague, the morbid changes are numerous, and differ more or less in different cases, is but natural, if the workings of the morbid process—embolism caused by the zoöglæa masses in the capillaries, are taken into consideration.

"Dr. Salmon's '*Bacterium suis*,' discovered by him in 1885, as a substitute for his '*micrococcus*,' has nothing whatever to do with swine plague. It is a septic germ, readily kills rabbits (cf. Bulletin of the Ohio Agric. Experiment Station), and causes septicæmia, but has no connection with the disease in question. It is not for me to

say where Dr. Salmon obtained it, or from where he may have imported it.

"If one wants to investigate the cause or causes of an infectious or contagious disease, it is one of the first requisites that he be practically acquainted with the disease itself and the workings of its morbid process; that he be endowed with a fair knowledge of physiological and pathological processes in general, and that he be unprejudiced, and have no axe to grind. One who lacks these necessary requirements will never arrive at reliable results.

"When Dr. Salmon commenced his investigation of swine plague, in 1878, the same time I did, the idea that swine plague might be a bacteritic disease never entered his head (cf. his first report in the Annual Report of the Commissioner of Agriculture for 1878). At any rate, his first report is perfectly silent on bacteria, and he does not mention them at all until two years later, in his report for 1880, after my reports had been published, and not until he had studied up Pasteur's writings.

"Yours respectfully,

"H. J. DETMERS.

"Ohio State University, Columbus, May 28, 1888."

In closing I wish to offer the record of the following autopsy for careful consideration:

On May 13, 1888, accompanied by Dr. George Roberts, a graduate of the medical department of the University of Pennsylvania, as well as Regent of the University of Nebraska, I visited a limited outbreak of swine plague upon the farm of the Hon. S. W. B., late a member of our state senate. It should be remarked that Dr. Roberts is quite an expert microscopist, and that in examinations and by cultivations he has found the same micro-organism as I have in cases of swine plague occurring where he lives—Creighton, Neb. He says "he cannot find the Salmon hog cholera microbe."

AUTOPSY XVI.:

Small black pig, three weeks old; yellowish, semi-fluid discharge from the anus; some few diffuse, bluish-red spots on the inferior surface of the abdomen. Animal killed in the laboratory by bleeding. Blood somewhat thick and of a bluish-red color. Abdominal cavity contained no effusion. Small intestine normal, but exceedingly contracted. Vessels of large intestine engorged, and a few petechial and some large, diffuse, hemorrhagic points in serosa of cæcum. Mesen-

teric glands some swollen, parenchyma lustrous. Peritoneum free from hemorrhagic centers. Stomach normal, contained some milk in a curdled condition. No changes in small intestine. Ileo-cæcal valve some swollen. Apex covered with a yellowish, caseous mass. Mucosa of large intestine somewhat swollen, but except for about one and a half inches in the colon, showed no signs of acute inflammation; here there was capillary redness with engorgement of some of the larger vessels. All through the cæcum and colon were to be seen numerous yellowish spots, from the size of the head of a small pin to three or four times that size; this yellow material was dry and friable, caseous, and could be easily removed, leaving small, irregularly-shaped cavities in the mucosa. Other changes were not present.

Kidneys: capsule non-adherent; outer surface and cortical substance opaque, swollen, anæmic, of a dull, yellowish-grey color; medullary hyperæmic.

Liver: swollen, as a whole, acini swollen, cut surface yellowish-red in color, and anæmic.

Spleen very much swollen.

Organs of chest normal, except the heart, the myocardium being opaque and anæmic, of a greyish-red color. Bronchial glands swollen.

The above case is of special interest, and is only introduced here because if there ever was a case of swine plague which absolutely corresponded, in every particular, to Mr. Salmon's "hog cholera," this one did. Hence the most exact search was made in seeking micro-organisms, but in the blood, in the spleen, in the liver, in the lymph-glands, both Dr. Roberts or myself could not find any other organism than that described by me in this report as the only germ of swine plague. Cultures were made and the same organism developed. Dr. Roberts being here with me, and interested in this work, as well as being a Regent of the University, he was doubly interested in endeavoring to find anything like the organism described by Mr. Salmon as occurring, according to him, in just such cases. Here was no room to say with Salmon, the lungs being affected, the germ of swine plague and that of hog cholera may both be present. After spending more than an hour in examining and re-examining the fresh organs, all of which were kept carefully covered, except for the moment necessary to clip out a small piece of tissue, we gave up the search, finding nothing but the one organism, and nothing resembling

in any way the one described by Mr. Salmon in 1885 as the cause of swine plague, which had no polar staining whatever, and in 1886 as the cause of his "hog cholera."

Such testimony as this should be conclusive. A mistake is absolutely impossible under the circumstances. This result simply corresponds with over 500 such experiences.

With this we close this side of our testimony, and trust that we have unquestionably shown that there is but one swine plague known in the United States, at present, and that that one is caused by the ovoid, belted germ, and that the lesions are due to general infection, and the disease an extra-organismal septicæmia.

PART III.

NATURE. SYMPTOMS. DIAGNOSIS. PREVENTION.

THE NATURE OF SWINE PLAGUE.

To discover the true character of natural phenomena is the chief mission of Science. The inner acts of Nature are largely hidden from our view. The final end of all scientific investigation is to arrive at an effect. This effect is not only the expression of the action of some unknown cause, but it veils that cause from our view. Behind the last veil we can never get, notwithstanding the subtle abilities of the developed brain, or the wonderful analytical power of the scalpel, the crucible, or the most acutely defining lense.

The mission of Science and limits of human endeavor can never be more truly and beautifully expressed than in the language which Edward Arnold has clothed some of the principles taught by Gautama, when he says:

“ Shall any gazer see with mortal eyes,
Or any searcher know by mortal mind,
Veil after veil will lift, but there must be
Veil upon veil behind.”

How historically true of the past, how prophetically true of the future. Once nothing was known of the structure of animal life, or of the actions of its elements, or the laws that control their action. All seemed hidden behind an impenetrable veil. The darkness must have seemed as crushing to the Aryan priests at the foot of the Himalayas as it now is to the uneducated and unreflecting, notwithstanding the evolutionary development of so many thousand years. “Veil upon veil” has been lifted, but the masses still refuse to gaze beyond the lifted curtain. Even to-day they know as little of themselves as those early Aryans who first gave us records of attempts at lifting the veil. How glorious the band of veil-lifters in our special branch of scientific research. Beginning with that majesty divine, Aristotle, what a flood of light was shed in behind the corner of the curtain lifted by this son of Grecian culture. So well was it lifted that even the clouds of the dark ages could not darken its life-giving rays. The dropped corner was again taken up by Hippocrates, Galen, and the veil-lifters

of that period, though they added but little to the light reflected by the work of the greater master. For centuries it burned like the vestal-fires on the altars of knowledge, sometimes it flickered to a dim spark, but again it burst forth under the skillful hand of a Vesalius, and once more the flame started by Aristotle began to send its warming and dispelling rays into the clouded ignorance of the priesthood and sanctuary. For the first time the human form actually became divine, for the son of inspiration had given the veil a mighty lift. This time the light was let in upon structure, but how the structure worked, what gave it life and action, what gave it vitality, was still a veiled subject, until another genius touched the already lifted curtain with magic wand, and lo! the streams began to move and man was told something of that wonderful current, vital with life's mystic energies, the blood, and Harvey inscribed his name on the uplifted fold, and physiology was born. And so went on the work, a Hunter taking hold of another corner and letting light into the terrible mysteries of disease, and Pathology was inaugurated as another branch of Science to be followed by Boerhaave, Haller, Bichat, Laennec, Goethe, Müller and an ever-increasing army of indefatigable and devoted workers, each of whom has lifted the veil somewhat; each of whom has made Goethe's immortal words, "*Mehr Licht*," the watchword of their lives. But this lifting of the veil was somewhat one-sided; the other corner was still draped in mourning, in token of the griefs of suffering ages. Now and then some mighty soul made an endeavor to let in the light, but the wail of suffering millions only too manifestly shew how little the veil had been lifted. But the veil-lifter came, as he ever will come, when human suffering has borne its cross too long, and the immortal Jenner gave it such a lift as man had not seen for many a century. Now both corners were up, but still the work went on. The veil-lifters increased. On the one side worked a Rokitansky, or a Virchow, on the other a Pettenkofer or a Koeh, while a Helmholtz, a Du Bois-Reymond or a Ludwig took hold of the middle, and then with one mighty throw a Pasteur lifts up the corner still hanging as Jenner left it, and again the suffering millions not only see "*Mehr Licht*," but so much light that they become uneasy in their hunger, and demand more and more, until a vast army of veil-lifters spring into being, and the curtain is lifted so high, and the deep recesses of darkness become so illuminated that humanity is becoming dazed, and

wondering men are saying, "What does all this mean?" but still goes up the cry, "Mehr Licht." But this is only mentioning a most insignificant portion of the veil-lifters, for every branch of science has had her devoted representatives in the light-giving work.

However much we may lift the veil, it leaves us ever in the presence of an effect behind which is some producing cause, which in its turn may be an effect. But human endeavor must ever stand impotent to correctly interpret the cause of a final effect! No matter how much deeper it may penetrate into the mysteries of nature, there will ever be "veil upon veil behind." The veil will never be entirely lifted, and it is well that it should be so, else all stimulus to human endeavor would cease, and the race gradually relapse again into the darkness of ignorance. The history of human development would then repeat itself, but in a retrograde direction. Though we start with an effect, still we start with that effect as a cause.

Let us illustrate this fact by the disease under consideration. Beginning with a dead hog as an effect, we make an autopsy, and come to other effects which in themselves have been causes in the death of the animal. What these effects are we need not touch upon here; we subject these macroscopical effects to the analysis of the microscope, and in the cells of the organs find those finer changes which have been the cause of the macroscopical lesions. So the work proceeds; until, aided by the life-inspiring genius of a Koch, we still follow these effects, and in the most of them we find—what? A bacterium! How wonderful the word looks to the uneducated mind! The very name, if mentioned in connection with water, ice, or food, seems to frighten half a community out of its wits. How much, on the other hand, it expresses to the competent pathologist! What a flood of light this discovery lets in upon hitherto unexplainable mysteries! How much such a discovery lifts the veil! Then comes the comparison of all that is seen with all that is known upon kindred subjects. All this has been the result of the analytical method. The final effect so longed for has been discovered! In one sense the investigator is satisfied. He knows that this final effect, this microscopic object, is but an effect; but what an effect! He knows that this effect is the cause of millions of dollars of loss to his country, and thereby much suffering to humanity. He also knows that this effect has a cause behind it that he may never discover. But does that discourage him? No!

No ! It only stimulates him to renewed endeavors to lift the veil, even though but a little more. His analytical studies have shown him that this minute effect is ponderous in its action as a cause. He commences to build ! Instead of the analytical method he has resort to the synthetical ! He would discover how this microscopic effect has produced that long line of effects over which he has traveled before he discovered it ? So he begins at the beginning. He has learned something as to the life-necessities of this effect by his studies of other effects of a similar nature. He knows something of the elements it needs to live upon. So he cultivates it. He watches how it grows. How it deports itself in different media. He inoculates healthy animals, and if his first effect has been the right cause, he again produces the line of effects and causes and causes and effects which he passed over in its discovery, and again produces the final effect, death ! So the veil is lifted ! So one victory is won ! But this last effect inspires him to retrace his steps somewhat, to less fatal effects. He returns to the sick animal and studies it as an effect, but also a cause in its relation to healthy animals of the same species. He studies these effects and causes in their relation to each other, and finally determines the nature of the diseased effects upon healthy organisms, and so light is let in on a dark question, and the suffering swine breeder finds that the disease is not contagious, but infectious, and has some comfort in that he feels he can see means to prevent his losses. And so from effect to cause and cause to effect the investigator goes on patiently and ceaselessly with his work, letting in the light, but still he finds other calls for his ambition. Still he finds

“ Veil upon veil behind.”

Such is the work of the pathologist ! His work is not only analytical and synthetical, it is more. After tracing all these effects and causes to the final effect, and from effect again, as primary cause, to the last fatal effect, death, he must now endeavor to explain *how* all these things have occurred and their direct relation to each other. The description of all these effects as they appear to the eye is comparatively easy work, but to interpret them correctly, and tell how they have all been produced until the primary effect is lost in the shadow of the veil, is the work of the master mind ; hence the pathologist must be a philosopher as well. The world sometimes calls him “ a theorist.” The half-educated and mercenary practitioner

crowds over him as a mere laboratory worker; "a visionary person without practical experience," yet but for this same "theorist," that very practical ignoramus would be almost without the means of making a livelihood. This "theorist" is the real lifter of the veil. The very "practical" practitioner is the dead weight which has to be continually overcome before the real value of the light gains entrance to the public mind. The correct interpretation of the "how" is the real problem in all research. The correct interpretation of the "how" is the first standard by which all work must be judged. Disease, then, is the summing up of the action of the effects upon the normal elements of the organisms. It is a disturbance of the normal action of these elements to such a degree as to threaten their continued existence. The nature of the primary effects, as well as cause, of the swine plague has been already portrayed in the section upon etiology. How this effect acts, what effects and causes it produces, until the final effect is reached, has been described in our treatment of the lesions. We have now to endeavor to shed some light upon the origin of the primary effect, and then upon the diseased organism as an effect in relation to those still healthy; and finally, how the primary effect departs itself within the organism infected as the cause of the lesions already described.

Having, therefore, learned something about what acts, and knowing what changes it has produced in the animal tissues acted upon, we have now to try and learn something as to how this action commenced, where it originated, and how it departed itself in the infected organism.

First then,

HOW AND WHERE DID OR DOES THE CAUSE OF SWINE PLAGUE ORIGINATE.

In other words, what is the nature of swine plague as to its origin. That is, is its specific nature

CONTAGIOUS OR INFECTIOUS?

This is the question of all questions to be decided properly, for unless we answer it correctly all our studies have been vain, and all that we have previously written is not worth the paper that has been used, not to speak of the labor and expense, except that it may represent a certain amount of labor saved to future investigators. The

correct interpretation of the facts that our detailed observations and experiments have brought to light is, however, the test by which our work must be judged. All that has gone before simply indicates diligence. That which we are now undertaking must show whether we have brains enough to value our experiences correctly. It is the philosophical inductions from scientific and practical experiments and observations upon which the world depends entirely for its advancement. Results obtained in this way are all there is practical in the world. That which the world calls "practical" has generally been paid for in vicarious blood money.

In my earlier communications upon the specific cause of this disease, the micro-organism, I took occasion to state the fact that the swine plague is an infectious and not a contagious disease, and that, in essence, it is a specific septicæmia of extra-organismal origin; that is, due to a specific micro-organismal element which finds its primary development and natural conditions for its vital support outside of the porcine organism.

From that which has been already said, the reader will easily see that the establishment of the true nature of swine plague is of infinitely more importance than the discovery of its specific cause, or even the discovery of a practical means of prevention by an artificial vaccine. Up to this time nearly all the investigators in this country have pronounced swine plague to be a contagious disease. This opinion is unequivocally wrong. Because it is wrong is the reason that all attempts at its prevention have so utterly failed. Swine breeders found that the results they attained did not correspond with the theoretic teachings. The interpretation was wrong.

Law says:

"This disease may be defined as a specific contagious fever of swine." Report of department of agriculture, 1878, p. 379.

Detmers says:

"Swine plague is a disease *sui-generis*, it is communicated from one animal to another by direct contagion and indirect infection." *Ibid.*, p. 332.

Salmon says:

"This disease is contagious, and in a great majority of cases may be traced to contagion." Report of 1880-81, p. 13.

"The demonstration of the contagiousness of the disease has enabled our agriculturists to do something to prevent its spread! Our investigations have shown that the swine plague is a non-recurrent fever." Report 1883, p. 57.

Leaving Professor Law entirely out of consideration, as his definition of the porcine pest can be equally well treated in connection with Mr. Salmon's, I will state that in a personal conversation with Dr. Detmers, as well as in his letter published in this report, he assures me that he agrees with me entirely that swine plague is an

EXTRA-ORGANISMAL INFECTION, AND NOT A CONTAGION.

My assertion that the disease is not contagious seems to have raised the ire of Mr. Salmon, for he says of it:

"Now what is the nature of hog cholera? Prof. Law and the writer have frequently referred to it as a contagious fever, but a recent author insists that it is not a contagious disease at all and that it is a strictly infectious disease. There is a good opportunity for hair splitting here, but let us not be beguiled by word-juggling when we have facts before us. Hog cholera is a bacterial disease; it is communicable from animal to animal by inoculation; when a diseased animal is introduced into a herd the malady rapidly progresses until nearly every animal in the herd becomes affected; the virus may be and is carried into all parts of the country by diseased animals. With these facts before us can we say the malady is strictly infectious and that any one who speaks of it as contagious is entirely wrong? It seems to me that such an assertion is supremely ridiculous and shows a lamentable ignorance of modern classification of disease.

"While writing this I have taken a few standard works from my shelves at a venture, and every one, including Ziegler's *Lehrbuch der pathologischen Anatomie* (4th ed.), Flügge's *Die Mikroorganismen* (2d ed.), Putz *Die Seuchen und Herde Krankheiten*, and Roll's *Thierseuchen*, include all bacterial diseases under the general term of 'Infectious diseases.' Ziegler then divides infectious diseases into miasmatic diseases, contagious diseases and miasmatic-contagious diseases. Flügge divides the infectious diseases according to the nature of the parasites causing them into contagious obligatory parasites, contagious facultative parasites, and non-contagious facultative parasites.

"It is not my intention to insist upon any classification in this connection. No student of pathology can be ignorant of the wide differences which exist between various standard authors. What I desire is to draw your attention from this never-ending controversy as to the

exact meaning which should be ascribed to the words infectious and contagious, and to concentrate it upon the facts in reference to this particular disease." *Journal of Comparative Medicine*, Vol. IX., p. 145.

Now that is all very wise, but the fact is that Mr. Salmon simply contented himself with making negative assertions, but never attempted to define what he thinks the difference between contagious and infectious diseases. Like the majority of the medical profession, he probably does not know. As to authorities, I cheerfully admit that he has them nearly, if not all, entirely upon his side. That does not make his assertions right, however. So-called "authority" has suffered terribly in the gradual advancement of our race. Facts are eternal! Authority transient! Facts are truths! Authorities are frequently mistaken interpreters! I care no more for authority, when the facts are against it, than for the whisperings of an evening breeze.

Sufficient evidence has already been given to show that the swine plague is not a contagious disease, and much of that evidence has been quoted from Mr. Salmon's writings. The trouble is, that exact observers and logical thinkers are just as rare phenomena in the medical profession as anywhere else in the human species. The action and advice of medical authors with reference to contagious diseases absolutely contradict their assertions, when its most advanced members come to write about them. Practically, they treat them as they are! Theoretically, they write about them as they are not! Practically, they tell us that diseased individuals are the only primary source of origin of contagious diseases. Theoretically, they treat of them as if they owed their genesis entirely to outside conditions, and utterly overlook their idiosyncratic origin.

As an example of this, I will quote from Mr. Salmon.

He says:

"Hog cholera is a bacterial disease; it is communicable from animal to animal by inoculation."

Robert Koch and the authorities quoted by Mr. Salmon (the veterinary ones aren't worth much), as well as hundreds of other equally reputable observers, say the same thing.

Hueppe says of the "Wild-seuche:"

"Experimentation has demonstrated that the natural eruption of the exanthematous form must be due to infection through accidental wounds in the skin of animals by the presence of the infecting organism in the earth, mud, water, or dust of their grazing places. It is a fact that this exanthematous or cutaneous form occurs as a purely contagious disease and is transmitted from animal to animal."

In the first part of the quotation he flatly contradicts the last part, and then endorses his contradiction again in the following:

"The exanthematous form is by no means so frequent, under natural conditions, as the pectoral, and the pathological phenomena go to show that the disease should be generally designated as an 'infectious pneumonia.'"

"Septicæmia" would be far more correct! The pneumonia is a secondary lesion! The reader will observe that I have nearly every authority against me, and yet I boldly say I am right, and trust to be able to convince him that I am.

The fact that any kind of disease can be induced in a healthy individual by inoculation from a diseased one has nothing whatever to do with its primary origin as it occurs under natural and every-day conditions.

Such a procedure simply shows that a given disease can be transmitted from diseased to healthy animals by accidental or artificial means in both contagious and infectious diseases. This procedure has no value whatever, so far as it can throw light upon the genetical nature of a disease. That it has been, mistakenly, assumed to give such light, has been one of the chief causes of all this misunderstanding and confusion about the true meaning of contagious and infectious, as these words bear reference to the origin of two entirely different classes of disease. The modern bacteriological school has done little more than add confusion to a previously very muddled intellectual condition. Let us see if we cannot bring some logical order out of this unfortunate chaos.

According to the teachings of the authorities, rabbit septicæmia should be a strictly contagious disease. It fulfills the requirements of their chief postulate. It is transmissible from infected to healthy rabbits by inoculation. In that regard it does not differ from the swine plague, glanders, syphilis, and a host of other diseases.

But in two essentials it entirely differs from any of them:

1st. It is an artificial disease entirely! It has never been known to occur as a natural disease of the long eared rodents. Its origin was accidental! Its germ was found in the dirty water of the filthiest of streams, by Gaffky. It has never been known to pass from artificially inoculated rabbits to healthy ones. Cohabitation of such with one another never induces it. Let us take another. Cholera is spoken of as a contagious disease, and yet when thousands of people died in Naples, in 1883-4, scarcely a physician or nurse fell as a sacrifice to their noble devotion. How different would have been the case in small-pox or spotted typhus but for the blessing conferred upon humanity by the immortal Jenner? Abdominal typhus (typhoid) breaks out in a house; we remove the people, sick and well, from such a house, and, with proper precautions, have no fears for the still uninfected members of the family. Even though such cases occur as that quoted by Hueppe in his first passages, it is absolutely no proof of contagion. It simply shows that traumata of the cutis may give the locus inoculationis to certain germs, if susceptible individuals are in infected localities. The last are dangerous to healthy organisms, not diseased individuals. It is simply an accident occurring under natural conditions which is the counterpart of experimental inoculation. When Hueppe says:

“The exanthematous form of “Wild-seuche” is by no means so frequent as the pectoral, and the pathological phenomena go to show that the disease should be generally designated as an infectious pneumonia,”

he demonstrates the correctness of my assertion, and shows that the exanthematous form is but accidental inoculation under natural conditions. All infectious diseases must be transmitted to healthy individuals by either artificial or accidental inoculation, if they ever occur under the apparent influences of cohabitation, unless all the animals are exposed to the dangers of a common cause. Cohabitation itself can never give an iota of aid in the extension of infectious diseases if the place inhabited is not infected. There must be some intermediate, inoculative aid present in such cases. Animals affected with the Wild-seuche will never extend the disease to others unless there is some factor present to convey the infection, in one way or another, from the former to the latter. Breathing the same air will never induce it, if the place itself is not a center of primary infection. Let us take anthrax, as the most striking example of this point.

Admitted that flies can convey anthrax from an infected animal to another by first biting the sick one and then the well one, its proboscis being soiled with the blood; admitted that an animal with wounds on its limbs, treading in the manure of a diseased one, can become infected with anthrax; admitted that a man with wounds on his hands can become infected from the blood or offal of such animals and die of malignant pustule, still that does not make anthrax a contagious disease, as all these authorities say. Under proper conditions and with the necessary care on the part of the attendants, that none of the hay or utensils of any kind to which diseased animals have access, can come in contact with the healthy ones, we often limit an outbreak of anthrax to the originally diseased animal or animals, though there may be many others in the same stable; they may breathe the same air, eat of food from the same mow, and drink from the same fountain, if the disease germs have not gained access to their body at the same time with the others we can prevent their becoming infected.

Several very striking examples of this point have come to my knowledge since I began investigations in Nebraska. In no less than three cases has one single case of anthrax occurred among a lot of cows in three different stables in midwinter. The nature of the disease in each case was determined by microscopic examinations, cultures, and the subsequent inoculation of small animals. A second case of infection did not occur in either stable.

A still more striking case occurred in the stables of the Standard Cattle Company at Ames, Neb., during the winter of 1888. One single steer, of some 800, died during the night, without evincing any signs of illness the day before. The blood was sent to me for examination. It was full of the organism which has been barely alluded to in this report, as of an unknown disease and belonging to the ovoid, belted group.

Another and quite extensive outbreak among cattle, due to the same germ, has been reported to me, and the material examined. The removal of the cattle from the feeding yards and change of feed stopped it at once. The extreme virulence of the organism has been tested on mice. At Ames no further losses have occurred.

However fatal to the infected individual, no one who is acquainted with the true nature of such a disease fears to come in contact with it. I would make autopsies on cholera patients from now "till the

crack of doom," without any anxiety, in a non-infected building that was properly cleansed and disinfected. How different would be one's feelings in case of the spotted typhus? In such a case a good life insurance would be likely to be prematurely realized upon by one's friends! How different in case of the small-pox, unless one was vaccinated? In the one case we fear the sick or dead individual. In the other the surroundings. In the first the removal of the diseased and its belongings, and good airing, will soon remove the danger. In the last we can almost neglect the sick individual if we take proper precautions as to the locality infected. In the case of small-pox we look to the clothing and bedding and airing the house. In the case of abdominal typhus, or cholera, we look to the discharges of the individual, and the surroundings. If the bedding is not soiled, and toilet carefully attended to, we care nothing for the individual.

The place of origin is the first essential, and the manner of extension the second, in deciding the question whether a given disease is contagious or infectious.

Inoculation has nothing whatever to do with it!

Those characterized by cutaneous eruptions, or characteristic neoplastic productions are, generally, easily inoculable from diseased to healthy individuals; the same lesions being produced thereby—small-pox, foot and mouth disease.

Those which are more or less organic in their compositions are seldom transmissible in this form by inoculation—contagious pleuropneumonia.

Of all medical authorities Pettenkofer seems to be about the only one who has appreciated the real point of differentiation as regards contagions and infections. He speaks of the former as endogenous and the latter as exogenous diseases. These two words have reference to the locus of primary origin only.

It matters not what preconceived ideas may have been; it matters not what the authorities may say, the one question is, what is the nature of a given disease, as to primary origin, in accordance with all known facts? From this, the only logical point of view,

SWINE PLAGUE IS AN INFECTIOUS AND NOT A CONTAGIOUS DISEASE.

What then is a contagious disease?

A contagious, or endogenous disease, is one which, so far as we can

historically trace it, finds its primary origin in the individuals of some given species of animal life (and never in any other way), and then passes directly from the diseased individual to another susceptible one either by direct contact or cohabitation, or by contact with some effluvia, secretion, or other material which has come directly from, or been in contact with such a diseased individual.

Transmission by inoculation has absolutely nothing to do with deciding this question! The locus of primary origin can alone decide it!

I boldly challenge the whole army of authorities to prove the above definition incorrect. They simply cannot!

It is so axiomatic that every layman who has been married and had his children go through the terrors of scarlet needs no other evidence to know that it is true and consistent with the facts. The lesser aggravations of mumps, measles, chicken-pox, etc., simply confirm a self-evident fact. The costly experiences of the country in glanders and pleuro-pneumonia confirm it in the minds of our stock breeders. The very rarity of rabies adds only more conclusive testimony to the fact that those authorities who assert that diseases are contagious because of their transmissibility by inoculation, are simply blind leaders of still blinder and automatic followers. It must first originate before it can be transmitted.

Let us again restate these facts, for it is absolutely necessary that the world comes to a definite understanding upon these important questions, and to no class of humanity more so than the army of suffering live stock breeders of our great and growing western states.

Although to the ordinary and non-technical reader the previous remarks may appear very fine reasoning, it is absolutely essential that the owners of swine understand these points, would they practically prevent the swine plague. A contagious disease, then, is an *exogenous* disease; that is, a disease which invariably finds its primary origin in a specific element—also a micro-organism—which invariably finds its proto-development within the body of a given animal species. It is communicated directly or indirectly from one animal to another of the same species. Syphilis, pleuro-pneumonia. On the other hand, there are contagious diseases having a more extended dispersion over the animal kingdom, though they find their primary development in a given species, but can extend by contact, wound, etc., infection to

some other species. To this class of contagious diseases belongs glanders, which finds its primary development in solipeds, but also accidentally, never primarily, may be extended to cats, dogs, guinea pigs, sheep, goats, man, and some other animals, but never to cattle. Rabies, small-pox, foot and mouth disease of cattle are others. Another distinguishing point between contagious diseases and strictly infectious ones, aside from the locus of origin, is that though the specific cause may retain its vitality, under favorable conditions, for some time outside of the animal organism, still it does not continue its development there, nor does it infect any outside materials. It remains attached to such elements only! Its danger soon ceases! It loses its virulent capacity and vitality! The contagious element of glanders does not retain its vitality for over forty days when bound on the nasal discharge or other effluvia from a glandered horse. Rinderpest does not hold over six weeks in a stable. To this fact is probably due our good fortune in not getting that plague among our cattle, rather than to any protective system that we have. With regard to the lung plague I cannot say how long the contagious elements will retain their vitality outside the bovine organism, but certainly not very long. It is singular that this pest should have engaged so much study with the least scientific results of almost any contagious animal disease. Notwithstanding all it has injured this country, our government has never made any proper scientific investigations looking towards its prevention. The lung plague is positively and practically preventable by artificial inoculation with a vaccine. The Hatch experiment station bill will, it is to be hoped, break the monopoly which the Bureau of Animal Industry at Washington has held over original investigations in this country, with regard to our animal diseases, and we may now hope to see a large number of independent workers in different parts of the country, each endeavoring to outdo one another and acting as controlling agents over the work of each other.

That the swine plague does not belong to the contagious class of diseases should be self-evident to the readers of this report if they have carefully followed the evidence collected in the consideration of its causes and the previous remarks.

That it can only arise from infected surroundings, and that sick swine, themselves, are the chief means of infecting and reinfecting such localities, should be very clear to every one, but to none so much

as the swine breeders of the West, who have paid so many thousands of dollars, one can truly say millions, for their costly experiences.

There are some very singular things about swine plague, but not more so than may be seen in any contagious or infectious disease. As has been mentioned in another place, we cannot tell why an eruption of a given contagious disease is very mild in one year and the next is excessively severe in the same locality. The same thing occurs in the local, land, infectious diseases. In anthrax we know that a cold, wet summer, with a high level of the ground water, will almost surely prevent the eruption of the disease in a locality that under the opposite conditions would be very dangerous. We also know that the disease seldom has the same malignity in the same locality in any two seasons. The same is true of the swine plague. Why the disease should have laid dormant in the pens for thoroughbred hogs at the college farm from May, 1885, to December, 1887, is something I cannot explain. Why after such a severe outbreak the germs seem, for a time, "to lose their grip," that is, to lose their power of infection, is something also incomprehensible. Why in the same outbreak some animals should die in 24 hours and others be ill ten or more days, is always a puzzle to farmers, but to the patho-bacteriologist easily understood, for we know the number of germs infecting must have been much greater in the first case. My idea, in such cases, is, that such hogs eat plentifully of the manure of the sick ones, or when the infection is by the respiratory tract, in some way find cause for aspirating a much greater quantity; hence, the proliferation in the animal must be numerically greater according to the number of organisms taken in in a short period. It often happens that the introduction of new hogs, which happen to be diseased, is followed by an eruption in a previously healthy herd within ten days. These are the cases which give grounds for the senseless hypothesis that swine plague is contagious. Such observers do not seem to think that, in all such cases, the infection is by the intestinal canal, and that the sick hogs, thus introduced, are soiling the food, refuse, and ground, and thus planting the inficiens where the healthy animals can most conveniently have access to it from their habits. In infectious diseases it is the natural excreta which are to be feared—anthrax, typhus, cholera, yellow fever, swine plague. In contagious diseases it is the breath, the effluvia, the clothing, the cutaneous desquamation, or secretions, but not the natural excreta which are the cause of danger.

Not one single fact can be adduced in the clinical history of swine plague which goes to show that it is a contagious disease.

In the report of the Department of Agriculture for 1883, Mr. Salmon told the suffering swine breeders of this country that, "the demonstration of the contagiousness of the disease (swine plague) has enabled our agriculturalists to do something to prevent its spread." I. e.

I challenge Mr. Salmon, or the Agricultural Department at Washington, to show one single fact, which has occurred since 1878, that will demonstrate where the work of that department, except Detmers', has been of any benefit, whatever, to the porcine interests of the United States?

On the contrary, I challenge them to show evidence that the erroneous doctrine, that the swine plague is a "contagious disease," which has been largely followed by our swine breeders, has not been a curse to the country, and one cause, not an unimportant one either, of the continued extension of the disease, and a source of unnecessary discomfiment to thousands of men?

I boldly say that if there had never been an Agricultural Department at Washington; if there had never been an investigation of our swine diseases made under the auspices of that department, save the forgotten ones of Dr. Detmers; if the present chief of the Bureau of Animal Industry had never been heard of at all, that the porcine interests of the United States would be no worse off than they are now; and, if erroneous doctrines, "unwarranted statements," and deceptions of the public count for anything in such questions, that this great interest, the greatest of all our live stock interests, would even be better off than at present.

Before defining what swine plague really is, I wish to point out another ridiculous and illogical statement of Mr. Salmon's as to the nature of this malady. He says it is a "contagious fever"! Bosh! No mortal human ever heard of "a contagious fever." Every disease is more or less of a "fever," and even where disease has not made itself apparent, fever is frequently present. *The fever* is a common accompaniment of every physiological disturbance. Fever, heat, cannot be transmitted even by inoculation! Hence, "a contagious fever" is a pathological impossibility! The causal element can, however, be transmitted both naturally and artificially in any contagious or infectious disease. Specific fevers have no existence except in the illogical

minds of non-reflecting authorities. There is the "authority" again! Wherever it elevates its offensive head against the hard logic of facts, decapitation is the natural result.

SWINE PLAGUE, THEN, IS AN INFECTIOUS DISEASE.

What, then, is an infectious disease? How does it distinguish itself from a contagious one?

In its primary origin only, so far as the point under discussion has relation to the disease.

An infectious or exogenous disease is one which invariably finds its primary origin not in, but outside of, an animal organism. That is, in the earth or in the surroundings of animal life where its microbic cause develops under certain conditions of the climate and soil which offer favorable climatic and telluric influences to its development. Such diseases are always local in their origin. The earth bears the same relation to infectious diseases that the animal organism does to contagious; that is, they both form the primary center of development in their respective class; but with this difference: the focus of primary infection is fixed in infectious diseases, while it is movable in contagious.

In contagious diseases the infected animals are movable centers of contagion; that is, wherever they go they carry with them danger to the infection of other susceptible animals directly from them. Now something somewhat similar occurs in infectious diseases. The infected animals may also become movable centers of infection; but in this they differ much from those diseased with a strictly contagious disease. They do not infect other animals directly, but they bear the same relation to the land that animals diseased with a contagious disease do to healthy susceptible animals. They can and do infect new land, or localities, and may thus be the means of extending an infectious disease over a broad extent of country, but only so long as they are actively diseased or have the infection within them, brought by them from its primary locality. On the other hand, they also re-infect the very localities whereon, or in, they became infected. In this way local centers of permanent infection are produced. This they do by means of their excretions. In this way anthrax, swine plague, southern cattle plague, the cholera, and yellow fever originate and become permanently located or are extended from one place to another, but no matter how much they may become extended they ever remain local diseases.

Now, while individuals thus complicated by an infectious disease may play an essential role in its extension, still there is this difference between the "seed," if I may be allowed the word, planted by them, and infected material dropped by and from an animal having a contagious disease. In an infectious disease the infecting material pollutes the earth, or local materials, and finding there its natural nutriment, if the climatic conditions are not unfavorable, it will retain its virulent activities for an indefinite period, and in many cases acquire a degree of virulence a part of which had been lost in the animal organism; notwithstanding the severity with which it acted in the same, it goes on multiplying indefinitely. In a contagious disease the circumstances are entirely different. Here, the animal organism forms the natural home of the inficiens, but when it is dropped outside of the animal organism, in some effluvia from it, it soon loses its virulent activity and does not multiply in the same manner as the inficiens of an infectious disease. Now any one can easily see that these are very essential points of difference, and that they distinguish these two classes of disease so sharply from one another that no one need be in danger of mistaking the one class for the other if they have sufficient education, observing powers, and logical acumen, and do not bow down in abject worship before authority.

Hence, swine plague is an infectious disease!

As I have said, by far the most important point which our studies of swine plague in Nebraska have established is that we have made this point clear.

It is an infectious disease and not a contagious one, as has been taught for the past ten years by the Agricultural Department at Washington, an error they have not yet been honest enough to correct, although every and all attempts at prevention must be based upon the definite settlement of this point, first, as measures of prevention which will be thoroughly successful in stamping out a contagious disease will be found futile against one of an infectious character.

In the former we have to seek the causes in diseased animals and their movements only. Stop them; kill off all diseased and exposed animals, and do it thoroughly, and the disease must be at an end. The supplying cause has been killed out, even though we know nothing of the specific one.

How different the ease with an infectious disease that finds its origin in certain conditions of the climate and land. Killing off and disinfection, which would be absolutely successful against contagious diseases, have but little or no value here. We cannot disinfect the broad hog runs of the West in any such easy manner. Were we to kill off every hog in the West and not have one in the whole country for a whole year, and then get healthy hogs and put them on the same land and in the same pens, in a very few years we should have as much swine plague as ever. Were we even to burn all the sheds and strawstacks, and put well hogs back on the same land, while the disease would not break out to any such extent as previously, still it would come again and gradually attain its original extension and devastating power, according to the conditions of temperature, moisture, etc., prevailing.

Hence, look out for the lands where diseased hogs have been. Hence, swine plague is an infectious disease pure and simple.

Something more than natural susceptibility and cohabitation is necessary to infection in strictly infectious diseases; that something is generally accidental infection, whether it be by the digestive or respiratory tracts or through cutaneous wounds.

The animals simply play the part of local conveyers or centers, offering favorable conditions for the intra-organismal development of the etiological moment, which never originates primarily in them.

Take for example the southern cattle plague, one of the most singularly striking of all infectious diseases, and one which departs itself in the exact manner of its twin sister, the yellow fever, as well as the swine plague.

A herd of Texans (infected on their native plains in the usually mild manner) is driven north. People do not notice any strikingly ill phenomena about them. They lie over in B's pasture, the next day in C's, and so on. Some weeks may pass by and B and C put their domestic cattle in the same pastures that the Texans were in. Their cattle die. Previously they were in the adjoining pastures; they may have smelt of and licked some of the Texans; the direction of the winds may have been such that they breathed the air more or less polluted by the Texans, and they remained well, but when they went to the pastures the Texans had grazed over they became infected with the southern cattle plague and began to die.

The Texas cattle, in themselves, were not dangerous to B's and C's cattle, but they had that in their droppings which found favorable conditions in the earth for its life and development, and this earth, these pastures, the roadsides where such Texans have been, thus became infected centers of danger to northern cattle.

There is still another class of infectious diseases which it is necessary that we define, because they are very apt to be confounded with the previous one. These are known, or should be, as the

MALARIAL-INFECTIOUS.

How does this class differ from the strictly infectious disease?

Both are local in their primary origin; but with this difference, the malarial-infectious diseases always remain local. In both classes a susceptible individual must be in, or upon, an infected locality in order to become infected; but, while in infectious diseases a diseased individual can carry the inficiens, and thereby be the means of infecting other localities that may never have been pestilential, the case is quite otherwise in the malarial-infectious; in which a diseased individual only becomes ill when living in such an infected locality, but has no power of infecting other localities, though he may leave the place where he became diseased and take up his residence in a place where it never has existed. Example: Fever and ague. They are both local, land diseases, nevertheless. In my opinion, the air, as a disperser of infection only, plays a far more important role in the infection of individuals in malarial-infectious than in the strictly infectious diseases.

Mr. Salmon quotes Ziegler as speaking of this class as "miasmatic-contagious" diseases.

I will simply ask the average citizen of the malarial districts of the South and West, did he ever hear of a person "catching" the "shakes" from one who had them?

Another manifest "decapitation" of authority!

THE NATURE OF SWINE PLAGUE AS DETERMINED BY THE INTRA-ORGANISMAL ACTION OF THE INFICIENS.

SWINE PLAGUE A SEPTICEMIA.

Let us see what Mr. Salmon has to say upon this interesting subject. In order to show that this gentleman could once see things as they

really are, and, himself, give the strongest evidence of the truth that swine plague is a "septicæmia," I must beg the reader's pardon for again using some quotations that have been equally useful in other directions.

In the U. S. Agricultural Report of 1878 Mr. Salmon asks the question,

ARE THESE LOSSES THE RESULT OF A SINGLE DISEASE?

And then answers it as follows:

"This question has been raised again and again, whenever any measure has been proposed for diminishing the death-rate of these animals; and, notwithstanding investigators in widely different localities have observed similar symptoms and similar *post-mortem* appearances, the objection to sanitary laws has always been the uncertainty in regard to the affection, or affections, from which death occurred. It therefore seemed advisable to visit a large part of the state (North Carolina) in order to decide this question of primary importance." p. 434.

Now what was Mr. Salmon's decision?

"Without exception the living animals presented similar symptoms, and the dead ones showed similar changes in the different organs of the body. Slight variations were of course observed, as is always the case in any disease, but these were as great between different individuals of the same herd, sick at the same time, as between different herds in different counties. And what is of great importance, I did not find a single case in which it could possibly be supposed that death resulted from a local disease. * * *

"Considering all these facts, there can be no doubt that these animals all died of a general disease, a disease not caused by changes in any single organ, but on the contrary a disease which causes the various organic changes observed." l. c., p. 435.

That is good pathology. Every word there written is strictly in accordance with the true facts. Now, in the name of logical common sense, I ask, what kind of a "general disease" could, or can, possibly have "caused the various organic changes observed" but a blood poison—a septicæmia?

It is needless to quote further from Mr. Salmon's reports prior to his last, for all through them he was of the "same opinion still." In his last report, as we have seen, he turns a complete intellectual hand-spring. Instead of a "general disease," he gives us two local diseases:

1st. An ulcerative-neoplastic enteritis.

2d. A "chronic pneumonia."

The reader would like to know how such cerebral "jugglery" came about? Now, a "juggler" is a clever trickster.

The reason is simply to be sought in the fact that I had outfooted Mr. Salmon and discovered the germ of swine plague, and that I had been fortunate enough to find, with Detmers, more pneumonia than ulcerative-neoplastic enteritis. Fortunately, or unfortunately, Salmon is a great worshiper of authority, and Shütz had come across three swine (only), out of a single outbreak, which were afflicted with the pneumonic side of the picture, and concluded that they represented the same disease his earlier examinations had to do with, (an error!) and therefore asserted the "*Schweineseuche*" to be a "pneumonia." This was an authoritative bolster to Mr. Salmon. He thought he had measure, though I had "knocked out" his forged germ of swine plague, 1885, he could now retain it by the forcible isolation of the intestinal lesions from the pulmonary. So he took the former to himself as an independent disease, "hog cholera," and left me the other as swine plague which he declared to be a pneumonia, for it will be remembered that in his report of 1886, he makes mention of some one having found a germ which was connected with pneumonic lesions, and probably the cause of them, or words to that effect, the exact ones have already been quoted. So, it is plain to be seen that Mr. Salmon is like the gambler who stakes his all on a forged note and then loses the game. He first forged a germ to save his hunger for priority and monopoly. Beaten at that, he goes back on his well-considered conclusion of 1878-1885 (when he had no opposition, and rightly considered the swine plague to be a "general disease," which of necessity must be a "septicæmia"), in order to still save that forgery, and against every principle of common sense, splits swine plague into two local instead of one "general disease." Hence, it is only to keep himself from sinking that he now grasps at the last plank, and, as a drowning sailor, tells his utterly incompetent audience that I—"the same author insists, in the most positive terms, that hog cholera is an extra-organismal-infectious septicæmia."

Reader, do not think I am fighting Mr. Salmon!

No! no! I am fighting for the truth, for the salvation of the great hog interests of the United States!

Then Mr. Salmon boldly eried for proof, knowing well there was no one in that audience who could either give it to him or contradict him.

The proof he wants is of two kinds:

1st. That swine plague is not a contagious, but an infectious disease.

We have partially answered that question, and will more conclusively show that it is not a "contagious fever" before we are done.

2d. That swine plague is a septicæmia.

That question Mr. Salmon answered for us in 1878, and gave us the same answer until 1886. We will, however, try and see if still more proof cannot be found.

Although, as intimated before, it would not make an iota of difference to me if all the authorities, living and dead, sacred and profane, were against me. I would face them all, did I believe—aye, did I know—as I do now, that all and every fact was on my side. Nothing is worthy a moment's respect but that which all the facts at command tell us is the truth. Science is the search for facts, for the truth—not the truth, but the search after it. Hence, to the scientist, the weight of authority often may be valueless. Truths, like good wines, improve with age. Authorities, like old bottles, often spoil the wine—i.e., the truth. The authorities, like the bottles, are finally "burst" by the advancing truth-seeker. Science has an army of fighters. Her standard is marked with the war-cry, "Seek truth." Truth is rankly anarehistic against authority unsupported by facts.

However, it is pleasant to have a little support, and Mr. Salmon has just a "leettle" in Professor Schütz, because, as said, he announced the "*Schweineseuche*" to be a "pneumonia;" *ergo*, "hog cholera" must be an enteritis! As I shall show, Schütz is on very shaky ground. The "*Wild-seuche*" is a "septicæmia," a general disease, but Schütz did not have acumen enough, or the weight of authority on Loeffler's part was too great to allow a difference of opinion or to permit Schütz to see that two similar germs could produce different effects, so he forced the "pneumonia" of the true swine plague into Loeffler's "*Schweineseuche*," which, again, Loeffler did not see was the "*Wild-seuche*," but Loeffler did see the disease he studied was a septicæmia, and I will show that Schütz contradicts himself, and is not certain of anything.

THE AUTHORITATIVE EVIDENCE THAT SWINE PLAGUE IS A SEPTICÆMIA.

Loeffler says :

“By the great importance which the diseases of swine have from an economic standpoint, their extended bacteriological investigation should soon bring us to definite conclusions, if the bacteria produce a disease belonging in the group of the erysipelas diseases, or if one is justified in looking upon these organisms as belonging to another specific disease of swine, viz., ‘Schweineseuche’ or ‘Schweine-septicæmia,’ and therefore to be distinguished from the genuine erysipelas of those animals.” Arbeiten a. d. Reichs Gesundheit Amt., l. c., p. 54.

Schütz says on this point :

“The previous experiments show that mice and rabbits which have been inoculated with small pieces of the spleen from a diseased swine become infected *or die of a septicæmia*, and that in the blood and tissues were to be found the same bacteria that infected the spleen of the hog, that is, the oval bacteria. Consequently it was proven that the spleen of hogs had pathogenetic action, and that the bacteria are the cause of this action. Hence their inoculation upon mice and rabbits *produces the same disease* as is produced by the direct inoculation of pieces of spleen from a diseased swine.” l. c., p. 383.

The reader will please observe that Schütz has said in the passage above quoted that the mice and rabbits which were inoculated with small pieces of spleen from diseased swine “*septicæmisch erkrankten und starben*,” that is, derive septicæmia and die therefrom, and again, “Rein kulturen fortgezuchteten Bacterien hatten, nach ihrer Verimpfung auf Mause und Kaninchen dieselbe Krankheit, hervorgeufen, wie die verimpften Milz-stücke,” which, rendered into English, is, that pure cultures of the bacteria had, when inoculated upon mice and rabbits, produced the same disease as pieces of the spleen of a swine that had recently died of Schweineseuche.

The necessity of presenting these facts from Schütz’s work will be self-evident when one reads the following words upon a later page of the same :

“Denn es steht nun mehr fest dass die durch die ovalen Bacterien bedingte, und als Schweineseuche bezeichnete Krankheit auch keine Septicæmia im eigentlichen sinne des Wortes, sondern eine infectiose Pneumonie ist.” Ibid., p. 402.

That is, it is now proven that the disease which is caused by the

oval bacteria, and known as swine plague, is not a septicæmia in the true sense of the word, but an infectious pneumonia.

Schütz seems to have forgotten that he had previously written that the disease produced in mice and rabbits by these same oval bacteria was a "septicæmia" and that it was the same disease as was produced by inoculations with small pieces of spleen of swine that had died of Schweineseuche.

If this is not a *contradictio ad absurdum* I do not know what is.

But even after writing the last passage quoted from the original, Schütz does not seem to be by any means sure of the correctness of his conclusion that swine plague is indeed an "infectious pneumonia," and not a "septicæmia" for he immediately qualifies that conclusion as follows :

"Notwithstanding, I prefer to hold to the name 'Schweineseuche' for the time being, as will be shown later, it is not proven to a certainty that the lungs are the only point by which the disease producing bacteria enter the porcine organism." Ibid., p. 402.

The conclusion of Schütz, that the "Schweineseuche" is an "infectious pneumonia," is most logically shown to be incorrect by Hueppe, though I think Hueppe mistaken when he includes Schütz's caseous-gangrenous pneumonia in his conception of the "Wild-seuche" simply because, as I have endeavored to show, the bacteria appear to be of the same species as those causing the "Wild-seuche." But, whether we accept the conclusion of Hueppe that such be the case, or follow my own idea that Schütz's second series of hogs represent the pulmonary lesions of the true swine plague, still Hueppe's criticism of Schütz is equally applicable to either disease, and shows that the primary lesion, the true disease, is a septicæmia and not a local, organic disease.

Hueppe says :

The experiments of Loeffler were considerably perfected through the demonstration, by Schütz, that the disease, also, frequently appeared in a pectoral form. Although the discoverer of this form (Schütz) treats it with considerable partiality, and has made public the idea, that the 'Schweineseuche' is not a septicæmia in any true sense, but an infectious pneumonia, still I must say that he has gone entirely too far in making such an assertion. As Johnes and I have shown, it depends, essentially, upon the modus of the infection, whether the 'Schweineseuche' appears as a septicæmia or an infectious organic disease." l. c., p. 776.

As I shall show, I cannot accept this last conclusion, although the entrance of the inficiens by way of the lungs may cause a bronchopneumonia, coeval with the blood infection, still every phenomenon of the disease shows that the pathological ens of either the swine plague or the "Wild-seuche" is a septicæmia and not a local organic complication. Were the latter the fact, then in cases where the pulmonary lesions were of so marked an initial character, those of septicæmia should not be concomitant with them, but follow after as secondary lesions, which is never the case.

Mr. Salmon might also find support for his latest assertion, that both his "hog cholera" and the swine plague are local organic diseases, in the following quotations from Schütz—where he says, with regard to the results of his second series of investigations:

"Upon the pneumonia follow the results of a general infection which takes place partly by way of the lymphatic and partly by the blood circulation. For the first speaks the severe changes of the lymph-glands, and for the infection of the blood the parenchymatous changes in the liver, spleen, and kidneys, and the irritation of the stomach and intestines." *l. c.*, p. 392.

As has been shown in another place, Professor Schütz most emphatically places the "cart before the horse" in any such reasoning.

This brings us to a point in the discussion in which it becomes necessary for us to know

WHAT IS MEANT BY THE WORD SEPTICÆMIA,

and then we shall be able to show how Professor Schütz is wrong in his conclusions.

First as to the word!

As I have no Greek type at my command, the radices of the word will have to be printed in Latin letters.

Septicæmia is from the Greek "*Saptos*," putrid, and *haima*, blood. That is, the addition of some putrid, disease-producing element (in solution) to the blood. It is very important that the meaning of the words "in solution," be fully understood. Bacteria, of themselves, in the blood do not constitute "septicæmia," for we may introduce many known forms of micro-organismal life without producing a septicæmic condition of the blood. We may even introduce germs known to be septicæmic, as to their product, into the blood of carniv-

ora—dogs—without even seeing the fatal effects of blood poisoning. It is the character of the material secreted by the micro-organisms and added to the blood which constitutes a given species septicæmia. The word was originally invented long before humanity had any idea of micro-organisms as a cause of septicæmia. The ancient authors observed that in cases of putrid ulcerations, or non-healing wounds, that a general disturbance of a peculiar character frequently resulted; hence, they assumed that the fluids of this putrefaction were somehow absorbed and distributed over the body, or, in other words, that like the character of the wound something putrid was taken up and constituted a part of the blood. Hence, the word “septicæmia!” It matters nothing to the etymology of the word whether that putrid something is generated by micro-organisms or not. Septicæmia can be induced without the presence of such organisms in the tissues.

We can free solutions, in which septic germs have been cultivated, from them, and by injecting the thoroughly germ-free fluid into a blood-vessel, produce temporary septicæmia. The same can be done with chemical derivates from putrid material. The difference between such a septicæmia and one due to micro-organisms in the blood is, that in the latter the amount of septic material is constantly being augmented. It can thus be seen that septicæmia can be easily classified into several forms according to the way they have been induced.

A doctor von Mansfelde, who has been known to say that he enjoyed the enviable reputation of being “the pathologist of Nebraska,” recently undertook to review my work upon the “southern cattle plague.” In the course of his remarks may be found the following passage:

“Septicæmia is not a specific disease, nor, as a matter of course, can a non-existing disease have a specific germ—septicæmia is simply a product of disease—varying from a mortifying stump of a limb to typhoid fever, pneumonia, and metro-peritonitis—Dr. Billings certainly knows this.”

“Dr. Billings” knows nothing of the kind!

The reader will remember the introductory remarks of this chapter as to effects being causes and causes effects. A person who cannot recognize this fact is certainly not a philosopher, and as pathology is the philosophy of disease, and the “doctor” fails to recognize this fact, it is axiomatic that he has no claim to the reputation of

being "the pathologist of Nebraska." He tells us that "septicæmia is not a specific disease." To that statement I would ask, what is the cause of septicæmia? Is it not a specific effect? Is it not in the southern cattle plague, swine plague, and such diseases a specific micro-organism? An effect of unknown causes, nevertheless a specific object? Now like begets like. If the cause is specific the effect must be also. Again, there can never be an effect without its possibly becoming a cause. Septicæmia is the specific effect of a specific cause acting upon the blood, and as it threatens the existence of certain elements of the blood, it must be a disease of the blood. We shall return to this point again. It has been shown that specific organisms, to which I have given the name of "ovoid, belted germs," are the cause of a large class of diseases, as well as that nearly every investigator has looked upon each of these diseases as a specific septicæmia. These diseases are, rabbit septicæmia, Wild-seuche, the southern cattle plague, yellow fever, and others of the same pathological character; but caused by bacilli, as anthrax, malignant œdema, emphysema infectiosum—black leg, etc. How, then, can the doctor, were he a pathologist, say, "nor as a matter of course can a non-existing disease have a specific cause?" It is perfectly evident that the honored gentleman does not know what disease is. Let us see what a septicæmia really is? Is it a disease or not? To answer that question we must first define disease!

Disease is any condition of any cell, tissue, organ, or organism which threatens the continued existence of that cell, tissue, organ, or organism. The blood is a tissue. It is composed of cells and intercellular substance—the plasma. It has been said that septicæmia is the addition of any septic material, in solution, to the blood. The plasma, being the fluid, intercellular tissue of the blood, it is evident that this septic element must be held in solution by the plasma. The cells float in this plasma, and experience has shown us that the red blood cells suffer in these septic diseases. Their existence is threatened! Hence, septicæmia must be a disease! The action of the septic-toxin upon the blood varies according to the specific character of the micro-organism producing it. It is this that makes them really specific in their effects. The blood lesion is the specific disease. This is something that a good many dilitanti observers and would-be pathologists have yet to learn. Sometimes this toxin is actually destructive to the red

blood cells, and the blood is not only thinner than normal, but is also more of a colored fluid than blood. This is the case in the southern cattle plague. In other diseases the toxin added to the blood by the specific micro-organism exerts more of a chemical action upon the red blood cells; it either destroys or materially overcomes their affinity for oxygen, while having less destructive action on the cells themselves. In such cases, anthrax, swine plague, the blood is very dark, blue-red in color, and thick, or, as it is often described, "tar-like" in consistency. These statements must be simply taken as describing the superficial lesions of the blood in septicæmia. Here, then, we have not only an effect upon the blood, but also an effect which becomes a cause of other effects, which, like it, constitute disease in other tissues.

To return to our reviewer, who again says:

"That all these diseases are accompanied by a great destruction of the tissues of the body, and that thereby a septic condition is invariably developed, no one can deny; but it is also true that the septic condition is the ultimate effect either of the decaying tissues of the organism, of a poison which may emanate from the bacteria, or as a product of their metabolism (Brieger's typhotoxin)."

In the above we have three different statements, which we will consider as they are mentioned.

1st. "That all these diseases are accompanied by a great destruction of the tissues of the body."

What causes the destruction? Was it innate in the tissues?

On the contrary, has not surgical experience taught us that if we can keep a given something away from injured or wounded tissues, that there is no danger of septicæmia. We know that that something is of a microbic character! Hence, the doctor's assertion, that "a non-existing disease can have no germ," is proven daily to be quite the contrary by his own acts, for I believe he justly enjoys the credit of being "the best ovariologist in Nebraska." Injured tissue can and does undergo a process of destruction, dissolution, and absorption in every case of traumatic healing without any danger of septicæmia, but only when we keep the germs away from lodgment in such tissues. No better example can be found than the contrasts between parturition when it runs its normal course, and septic metritis.

2d. The doctor tells us, "that the septic condition is the ultimate effect of the decaying tissues of the organism."

Nonsense!

The answer to that question has been given above, "keep away the germs, and dissolution, resolution, and healing will follow without septicæmia resulting." The germs cause the "ultimate effect"—the septicæmia.

3d. The doctor confirms every word I have said when he says, "that the septic condition is the ultimate effect * * of a poison which may emanate from the bacteria," hence the tissues having nothing to do with it. The non-surgically treated wound makes the atrium, however. Dr. von Mansfelde's whole fabric, therefore, has collapsed! He knows, or should know, that it is the law of good surgery: no germs, no wound septicæmia. The great master of anti-septic surgery, Lister, the savior to carry the balm of salvation into our pest-stricken, gangrene-producing hospitals, taught that principle, and every surgeon in the world acts upon it to-day, though each may have his own peculiar ideas as to the best method of carrying it into effect. Septicæmia is not a "product of disease," but it is disease itself. It is an effect and a cause at the same time. It is a product of germ life! But it is more! It is not only a product of germ life, a something added to the blood which induces disease of that tissue, but it again produces disease by its action upon the caloric centers. It produces fever. Fever, however, is not a disease. It is the product of the irritation of the septic material. It is, in a septicæmic disease, an intermediate factor between the blood lesion and those disturbances of the parenchymatous organs which it causes. In septicæmia, the fever is the transmitter of the primary action of germs, the blood disease, to the various organs of the body where the secondary lesions occur.

The same reviewer also asks some interesting questions, which are as follows, and though answered in another part of this report, it certainly will not be out of place, and also a courteous act to answer them here again. He says:

"Why Dr. Billings should coin a new genus for his southern cattle plague germ is quite incomprehensible to the reviewer—and how he comes to call it a belted, septicæmic germ, and the disease an extra-organismal, local, land septicæmia, he cannot understand at all. It

cannot be denied that the germ, if it is the cause of all the diseases mentioned, belongs to the same genus as the micro-bacterium of typhoid fever."

This question bears on its face the evidence that the doctor does not know anything, practically, of the subject upon which he is treating. First, let me tell him that the germ he speaks of has no resemblance to "the micro-bacterium of typhoid fever," other than some points of correspondence in development in artificial media. The micro-organism of typhoid fever is a bacillus and not a bacterium, but what distinguishes it far more effectually from the micro-organism of the southern cattle plague is, that typhoid fever is a disease of man, and, though local in its origin, extends to all climates, while the southern cattle plague is a local disease dependent upon certain climatic conditions for the development of its germ. Why did not the learned author say that the germ of yellow fever belongs to the same genus as the micro-organism of the typhoid fever, for I have said that it belongs to the same class as that of the southern cattle plague, and is far more like it than that of typhus abdominalis?

The doctor is again troubled to know why I have called the class of germs to which those of the southern cattle plague, yellow fever, and swine plague belong "belled," or better, "ovoid, belled germs"?

That question is easily answered, and I have no doubt that every patho-bacteriologist will thank me for such an attempt at classification.

Because we know of a large number of diseases, each of which have this in common, that their micro-etiological organisms all have striking morphological and cultivation-biological resemblances. They all look more or less alike under the microscope, and develop more or less alike in different media. They all have the same form and the same general staining reaction. But in one thing each is different from the other—they occur in different forms of animal life under natural conditions, some of them being distinctly limited to one species, as the swine plague, others occurring in several, as the "Wild-seuche," which attacks deer, bovines, and swine, and still another is not known to be a natural infection—rabbit septicæmia.

But, again, they all have one attribute in common.

They all produce septicæmia, and each one produces a specific septicæmia. Each one also finds a specific something in the species of animals it infects, which helps determine not only the character of

the septicæmia—its lesions—but also the character of the secondary lesions—swine plague.

Now were these the only varieties of septicæmia known, there would be no need of creating a special class for these micro-organisms. There are, however, several other varieties of septicæmia which are caused by entirely different organisms. For instance, we have anthrax, black-leg, and malignant œdema, caused by different members of the bacillus group, which could be equally well termed the bacillary septicæmia. More complete justification of my endeavor to differentiate this group of septicæmic diseases (from those caused by bacilli) by distinguishing the class of micro-organisms, which act as their specific causes, as “ovoid, belted germs,” than has been given by Hueppe, could not be desired. In speaking of the “Wild-seuche” (which, as I have shown, is very intimately allied to the swine plague, and caused by a micro-organism belonging to this “ovoid, belted group”), Hueppe says:

“This disease has undoubtedly been classified as one of the forms of anthrax in the past, and as Bollinger says, especially as gloss anthrax, white anthrax, or as anthracoid-pneumonia. Its resemblance to anthrax is very remarkable; especially in its clinical and patho-anatomical phenomena. We meet with the same carbuncular, erysipelatous form, the hemorrhagic intestinal mycosis, and the same hemorrhagic conditions in the internal organs which have always been described among the characteristic lesions of anthrax. The only reliable point of differentiation between the ‘Wild-seuche’ and anthrax is to be sought in the morphology and biology of the respective micro-organisms” (l. e., p. 776), the former being caused by an “ovoid, belted germ,” the latter by the well-known bacillus.

The so-called “surgical septicæmiæ” are largely due to micrococci. To repeat, we have, then, the still larger group of very distinct diseases—rabbit septicæmia, swine plague, “Wild-seuche,” yellow fever, southern cattle plague, and undoubtedly others, all caused by a different micro-organism, which is ovoid, colors only at its ends, and has round ends, and is marked by a colorless belt across the middle of the body. To distinguish this well-known group of micro-organisms I have coined the name “ovoid, belted germs,” so that when other observers discover new members of the same group, by using the term “ovoid, belted germs” we shall at once be made aware of their chief characteristics under the microscope, and much about their manner of development.

So far as we now know, we may also be justified in concluding that the disease in which such a germ is discovered is also a septicæmia. The word "bacillus" is somewhat expressive, but "bacterium" is not. An object belonging to the latter class simply has to be ovoid—it may be "belted" or not, as the case may be—so I thought, and still think, it right to have coined the term "ovoid, belted germs."

The doctor again finds fault with my having coined another term, "extra-organismal septicæmiæ." This was done for similar reasons, viz., to indicate that the organism infected became so from the organism infecting finding its place of primary development in the outside surroundings of the former. The locus of primary origin alone decides this point. Of this attempt to generalize a certain class of diseases, the doctor says:

"Why then manufacture a name for his germ or for his disease, or diseases, which can have no scientific basis? The typhoid bacillus will do as well for his southern cattle plague germ as the inclusion of the disease into the group of acute infectious diseases. This is so much the more to be recommended, since Dr. Billings knows that the germ of typhoid fever rejuvenates itself, multiplies in the soil, and that the outbreaks of typhoid fever and those of the southern cattle plague show a great similarity; not to speak at all of the very similar lesions, which are produced in the body of man and cattle by typhoid fever and the southern cattle plague."

The above is almost word for word unmitigated nonsense!

I think the term "extra-organismal septicæmiæ" has a most practical and scientific basis. It not only defines the nature of the disease, but tells us that the infection was from the surroundings of the organism infected; and when we add to that, by an "ovoid, belted-germ," we have told a long story in very few words. The doctor admits the correctness of the term "extra-organismal septicæmia" when he says that, "Dr. Billings knows that the germ of typhoid fever rejuvenates itself, and multiplies in the soil," and "Dr. Billings also knows that every known species of these pathogenetic, "ovoid, belted germs" also does the same thing. Hence, he thinks the term "extra-organismal septicæmia" was even more appropriate to the circumstances, thanks to the doctor's criticism, than he ever did before. There is one thing, however, that Dr. Billings knows that his estimable critique does not, and that is, that, except in the fact that their respective micro-organisms find their locus of primary development outside the

animal body, and hence, genetically speaking, are to the respective species "extra-organismal," and again that a "septicæmia" results in each case, that there is no more relation between typhoid fever in man and the southern cattle plague, or swine plague, than between a man and a gorilla. They belong genetically to the same great class of diseases; that is all. But, on the other hand, we have still another class to add to our septicæmic group. This class has but one representative which I call to mind at present. It is an absolutely intra-organismal septicæmia. It is due to the accumulation in the blood of used-up material that should be excreted by the kidneys, and is known as uræmia. It might be equally well termed septo-uræmia, but that would be unnecessary. Hence, the septicæmiæ may be logically and pathologically classed into three groups, viz.:

1. *Wound-infection septicæmia*, or surgical septicæmia. The wound and the surgeon's carelessness being the primary cause, while the germ is the secondary, the specific cause.
2. *Extra-organismal septicæmia*, due to a specific physiological idiosyncrasy of the species infected, as a primary cause, and the specific germ as the secondary or completing cause.
3. *Intra-organismal septicæmia*, due to retention of non-excreted material in the blood from the non-action of specific excretory organs—uræmia.

THE SWINE PLAGUE A SEPTICÆMIA.

It seems to me that no one can doubt that the swine plague is a septicæmia, a "general disease," as Mr. Salmon most truthfully said in his report of 1878, and not a "disease caused by changes in any single organ, but, on the contrary, a disease which causes the various organic changes observed." l. c.

Before considering this question in the light shed upon it by the patho-anatomical lesions of the swine plague, let us first give a moment's attention to the collateral testimony.

I think that I have sufficiently established the correctness of my endeavor to differentiate this group of septicæmic diseases from those caused by bacilli, by calling their micro-etiological organisms "ovoid, belted"germs. The fact that these ovoid, belted germs are the causal moment in these diseases is too well established to be questioned, so far as those herein mentioned are concerned. Now we know that one

of them, at least, was given the name of "septicæmia" by Gaffky from the outset, and that the name was selected because the character of the lesions so completely corresponded with those met with in other known forms of septicæmia. Hence, though he discovered the micro-organism alluded to in the filthy water of the Panke, and not in any infected animal, and then, by experiment, found rabbits were susceptible to it, he called the disease "Kaninchen-septicæmia."

The history and character of Gaffky's results and conclusions also scores a point for me, which, so far as this "Kaninchen-septicæmia" is concerned, shows it can be nothing else than "extra-organismal."

To repeat: As the disease was not known to exist in rabbits, and is not known to occur in them as a natural infection, and as Gaffky accidentally found the germ in the earth, and then, for the first time, induced the disease in rabbits, there is no room for argument that the Gaffky rabbit septicæmia was and is not of extra-organismal origin. Again, we have seen that men of undoubted ability, who have studied other diseases of this same class caused by these "ovoid, belted germs," and whose names are connected with the discovery of the specific germs—contrary to the reasoning of the talented master of pathology in Nebraska, Dr. v. Mansfelde—have also come to the same conclusion, that such diseases are also septicæmia, and even Cornil and Chantemesse admit the same thing as to the swine plague in France, though they mistakenly persist in calling it a "contagious pneumonia," when they say:

"The lesions described show that the contagious pneumonia is a general infectious disease, [they fail to see that in coming to this conclusion they flatly contradict their assertion that the disease is a "contagious pneumonia," for the pneumonia is an accidental, secondary complication!] rather than a local pulmonary affection."

This then should show us that the swine plague is a "general disease," and as the "Wild-seuche," and the "rabbit septicæmia" have been shown to be septicæmia, and as the yellow fever and southern cattle plague were known to be such long before the germ of either of them was discovered to belong to this same group, we may feel perfectly safe in concluding that the swine plague is also what Mr. Salmon said it was in 1878, and what Cornil and Chantemesse agree in saying it is—a "general disease;" and that I was right, in 1886, when I also said the swine plague is septicæmia, though to save his poor, unfortu-

nate, manufactured organism, and to try and be original, Mr. Salmon subsequently made the ridiculous attempt of differentiating swine plague into two local diseases. We have, also, called attention to the fact that it was because Schütz said that the German "Schweine-seuche" is an "infectious pneumonia," a local organic, instead of a general disease, that Salmon felt he had support in his absolutely unwarranted attempt to differentiate the one American swine plague into two essentially idiosyncratic complications.

SWINE PLAGUE AN EXTRA-ORGANISMAL SEPTICÆMIA.

Having, it is hoped, conclusively shown that this porcine pest is nothing more nor less than a septicæmia, by collateral testimony, and that of Mr. Salmon and other observers, it now remains for us to discuss the nature of the disease as to its genesis. But little need to be said upon this point, however, as by reference to our consideration of the etiology of swine plague, and the previous discussion the reader will find an abundance of incontrovertible evidence that swine plague does not belong to the—

1. Surgical septicæmiæ; nor to the
2. Intra-organismal, but to the
3. Extra-organismal septicæmiæ; that is, it is a disease, the genesis of which does not necessarily depend upon the pre-existence of traumatic lesions in the skin, nor upon the non-action of any of the organs of the body; but, primarily, upon certain unknown physio-chemical idiosyncrasies of the porcine organism, in health, and the entrance of a specific something into that organism, from outside, where it finds peculiar conditions favorable to its development and where through a specific physiological attribute it causes disease by the production of a poison which is given off in the blood and taken up in solution by that tissue, and then conveyed to all parts of the body; hence, is an extra-organismal septicæmia.

I think no further argument is necessary to show that this *iniciens* does not, at first, develop in the porcine organism and then pass from hog to hog; that is, that the disease is contagious. Every form of septicæmia, except, uræmia, whether due to trauma of surgical origin, or such as occur in the natural course of events, parturition, is non-contagious in origin, and non-contagious in extension; (but not non-transmissible by inoculation in the experimental sense of that term). In

every case, no matter how or where, the micro-organism, the cause of the specific septiceæmiæ, finds its locus of primary development, its locus of natural life, its only permanent habitat, outside of the animal organism. Its home within the infected organism is but transient! A temporary boarding place!

That the swine plague is an "extra-organismal septicæmia" need not be further discussed at present; as, unfortunately for the reader, the whole practical side of the question has got to be threshed over again when we come to treat upon prevention.

We can now bring this part of our discussion to a close by the consideration of

SWINE PLAGUE A SEPTICÆMIA AS SHOWN BY ITS LESIONS.

It would seem that the competent reader must have come to the same conclusion, by this time, to which I came with regard to the swine plague long before it was possible for me to fully comprehend, far less correctly interpret, its many peculiarities, and that is, that it is not only a very interesting, but also one of the most complex diseases of animal life. The uncertainty existing in the minds of European investigators, and their mistaken conclusions as to its nature and its lesions, are not to be wondered at when we consider how little actual experience they have had with it. While its specific micro-organism and the detailed character of the patho-anatomical lesions of this porcine pest may be all studied in the laboratory, I must say that no man can ever learn very much about the swine plague in that way. It must be observed most critically in the field, and by a very large number of hogs, at all seasons of the year and under every conceivable form of treatment of the animals by their owners, before any man is competent to give conclusive opinions upon this malady, no matter how fitted he may be in other respects to enter upon such investigations. To come to any trustworthy conclusions, as Schütz did, by the examination of six or seven cadavers, with a most imperfect history, and absolutely no clinical experience, even though a few experiments were made, can not only be misleading to the investigator, but be the cause of his innocently misleading others. It is to be hoped, as well as it is my sincere and determined purpose, that this work shall lead to the rapid solution of all the questions here discussed, if not settled by it, and most especially, that absolute justice shall be rendered to every investigator according to the character of his work.

We have now to explain the action of our primary effect, the bacterium, as the specific, or sufficient cause of swine plague. The first act in this drama is the entrance of this specific organism into the body of the animal affected. No matter how this entrance is effected, the primary result is invariably the same, viz.: the infection, pollution, of the blood. The production of septicæmia. This point need scarcely be emphasized again, but when so good an observer as Schütz falls into the error of looking upon pneumonia, in such a disease as this, as the primary lesion, one scarcely knows what to say. To one who knows him as well as I do, it seems absolutely incomprehensible. The neuroscopical notes introduced in order to show how the lesions in rabbits inoculated with pure cultivations of the swine plague germ differed from the results obtained by Loeffler and Schütz, the former especially, are sufficient evidence that the real effect of the germ is to produce a septicæmia. The more virulent the material used, the more acute the attack, the more is this fact apparent, as is also well shown by some of the notes made from autopsies upon swine.

Before going further I desire to call attention to an especial point that has occurred to me by which the specific action of the etiological organisms seem to differentiate the contagious from infectious diseases. The following remarks must be taken in a suggestive rather than an axiomatic sense:

CONTAGIOUS DISEASES MORE SPECIFIC IN THEIR LESIONS THAN INFECTIONOUS.

In endeavoring to establish a more practical and scientific meaning for the words contagious and infectious than has heretofore been the case, that is, making them apply to the origin—genesis of the inficiens in either class of disease—it is my hope that a great step has been taken towards clearing up the miserable uncertainty which exists in our medical literature upon the subject. Again, it appears to me that the contagious diseases differentiate themselves from the infectious with almost equal sharpness by the action of the inficiens. In those of a strictly contagious nature we generally find the inficiens manifests its specific character, aside from its genesis, by some almost pathognomonic peculiarity of the lesions in each disease. The idiosyncrasy of the causa sufficiens in contagious diseases may be either some peculiar cutaneous eruptions, or lesions, spotted typhus, syphilis, tuber-

culosis, scarlet, glanders, the foot and mouth disease, or by local action in certain organs, as in the mumps and contagious pleuropneumonia; while there are still others in which the specific organic affinity, on the part of the *infiens*, is well marked, still, so far as we yet know, pathognomonic lesions are entirely absent, of which rabies is a marked illustration!

Septicæmia as a primary lesion never occurs in contagious diseases. The blood simply acts as a conveyer of the specific germs over the body and as nutritive pabulum. The fever is the result of the direct irritation upon the tissues, rather than of a poisonous septic irritant in the blood. When septicæmia occurs it must be looked upon as a secondary lesion, or complication.

In the extra-organismal septicæmiæ quite the contrary is the case. Here the blood lesion and the sequential parenchymatous changes are the specific or primary lesions, while all organic are of a secondary or accidental nature. Even those which are more or less characteristic in their nature, such as the "enorm œdem" of Wild-seuche, the peculiar muscular complications of the black-leg, the erysipelatous redness of rouget, while pathognomonic to an essential degree, must be looked upon as deuteropathic manifestations, and not due to protopathic action on the part of the *infiens*. Pneumonic and intestinal lesions in these diseases must not be looked upon as essentially specific. They either indicate that the *causa sufficiens* gained access in one of these directions, or are secondary complications. Anything pathognomonic in these lesions must be attributed, far more, to something specific in anatomical structure, or to physiological idiosyncrasies in the species infected rather than to any primary action on the part of the micro-organisms infecting. Typhus abdominalis, swine plague.

Cholera, though a strictly infectious disease, is not septicæmic. It can be likened to those contagious diseases in which the *causa sufficiens* manifests its specific action in certain organic complications. The intestinal tract gives the locus of specific primary irritation in cholera.

Were the mentioned pathological complications due to the primary action of the *infiens*, such as the neoplastic products in the large intestine in swine plague, and not caused, in the first place, by peculiar anatomical or physiological conditions in the organism infected, we should be able to produce them also in rabbits, etc., when used for

experimental infection with the same germ. That we cannot produce such lesions is sure proof that such diseases are septicæmiæ. It will be remembered that as early as 1876 Klein pointed to this fact as proof against the conclusions of earlier observers that the swine plague could not possibly be typhus abdominalis.

Seeking such lesions, and missing them, in the smaller animals used for experimental inoculation, has frequently blinded observers from seeing the true pathological ens of these septicæmiæ. Hence, in animals in which such diseases naturally occur these ontogenetic lesions are often of peculiar diagnostic value, but at the same time their occasional absence has no negative worth.

One fact stands very prominent before us as to the patho-anatomy of the "Wild-seuche," which is this. Although known to investigators ever since Bollinger introduced it to the world as an idiopathic malady, and studied particularly by the professors attached to the Munich Veterinary School, among whom may be especially mentioned, Frank, Kitt, and Friedberger, and since Bollinger's time differentiated from anthrax; still, not one of these observers has ever mentioned, or intimated, the presence of any such lesions in the lungs of swine which have perished from that disease, as Schütz describes in his second series of examinations of caseous or gangrenous pneumonia, nor does one of them mention the so-called "characteristic" lesions in the intestines, as described by Roloff, and which are well known to occur in the swine plague.

Coming back, then, to our starting point,

THE ACTION OF THE BACTERIA IN SWINE PLAGUE.

The first thing that occurs after they enter the porcine organism is multiplication in the blood, and the giving off to the same of a specific toxic principle, which at once acts upon the red blood-cells and the caloric and respiratory centers, and causes an excessive rise in temperature and more or less disturbances in the circulation and respiratory functions. Now we will suppose that our animal dies during this stage of the disease; that is, in the course of 24 to 36 hours after the first attention is drawn to its being ill. What shall we find? Nothing but the phenomena of acute and excessive blood poisoning, and those due to the above named disturbances and the initial results of fever, of which the autopsy upon the college farm

hog that died Dec. 12, 1887, is a most striking example! What did we have there? A thick, dark, blue-red blood, almost black, of tar-like consistency, so much so that even in anthrax it would have been looked upon as extreme. Numerous hemorrhages in the serous and mucous membranes; swelling of the parenchymatous organs, with much engorgement and the beginning of inflammatory changes; engorgement of the lungs, but no inflammatory lesions. But, aside from the blood, the most marked lesion was the swollen and hemorrhagic condition of the lymph-glands.

THE PULMONARY LESIONS IN SWINE PLAGUE.

The greatest misconception exists as to the relation of these lesions to this porcine pest. The same is true of the Wild-seuche, according to the literature of German writers. In neither disease are the pulmonary lesions absolutely specific. While in many cases they may represent a primary lesion, in a certain sense, still they are not the specific, and even when so apparently primary in their date of inception, it is questionable, even then, if the blood poisoning does not actually antedate the proliferation which marks the genesis of the broncho-pneumonic centers. Where broncho-pneumonia is present in swine plague, or any septicæmic disease, it is simply a guide-post placed there by the germs to indicate to the observer that by these doors we entered in. From here they enter the circulation. From here the real disease begins.

In swine plague the broncho-pneumonia is simply due to the mechanical irritation of the bacteria as they pass from the delicate termination of the tubes, or accumulate at these points. That such material is caseous, that it fills the tubes at this point, is no such wonderful thing as Klein, and Salmon imitating him, point out. It is but the natural result of rapid epithelial proliferation, and as the lining of the adjacent air-cells is of the same kind, and the irritation at once extends to them, it is also natural that the product must be of the same nature, and that it should become caseous, for rapidly produced cellular elements are struck with death about as soon as they are born. The more rapid their reproduction the more rapid and complete the necrobiosis—hence, caseation. Schütz fell into the most unaccountable and absurd error when he looked upon the caseous broncho-pneumonia and the gangrenous, destructive processes in the lungs, in swine

plague, as having the same origin. He reasoned from phthisis in man. Admitting, most cheerfully, that Schütz is right when he says that the caseous broncho-pneumonia indicates infection via the respiratory tract, still the gangrenous processes do not find their origin in the extension of the broncho-pneumonia and complete filling up of the lobuli from this source in any acute case. The course of the disease is too short for destruction to arise from this source. I will cheerfully admit that such may occur as a sequel, but in over 500 autopsies I have never yet seen that sequel. My opinion is, that if swine can withstand the septicæmia and its sequential lesions, that caseous broncho-pneumonia will never kill them. The destructive lesions in the lungs in swine plague have an entirely different genesis.

They may or may not be caseous; they may or may not be complicated by broncho-pneumonia; the latter will depend entirely whether the inficiens entered the infected organs by the respiratory tract or not. They will invariably be lobular from the moment of their inception. In only very rare cases will the infiltration of the lobuli be dense, and hence, a dry, caseous surface will scarcely ever be seen. When the infection has taken place entirely by the digestive tract, or by means of intra-abdominal or subcutaneous inoculation, broncho-pneumonia will be conspicuous by its absence.

THE GENESIS OF DESTRUCTIVE PNEUMONIA IN SWINE PLAGUE.

We have already considered the specific action of the bacteria of swine plague in producing septicæmia, and spoken of the direct effects of the same, through fever, upon the parenchymatous organs. Among the latter the myocardium soon suffers very severely and becomes anæmic, yellowish-grey-red in color, and very friable. In fact, I have seen it so badly degenerated that it mashed up under the fingers like any moist, granular substance. Whether or not the toxin acts upon the intra-cardial ganglia, I do not know. It certainly does not upon the respiratory centers in the medulla, nor upon those of circulation, except as the excessive rise in temperature influences them, especially the latter, by general disturbances of the circulation. Naturally the disturbances lead to hyperæmic conditions in the lungs, and especially in those parts the least favorable to the active and regular flow of the blood. Here, stagnation and stoppage of the circulation soon occurs, according to acuteness with which the septicæmia

and its sequential lesions are developed; which is soon followed by disturbances and finally destruction in the vascular walls, and coagulation of the blood in those vessels soon follows, which is aided and abetted by the presence of innumerable bacilli, around which, as foreign bodies, coagulation also takes place.*

The large interlobular vessels, in such localities, will be engorged, of a dark red color, filling up the entire interlobular spaces of the complicated sections. (See Plate XII.) The neighboring lobuli will be at first hyperæmic or hemorrhagic, then œdematous, the same of the interlobular tissue, next more or less cellular proliferation occurs, but into a previously œdematous tissue; the more distantly situated and not so severely complicated lobuli will be less œdematous and more cellular in the character of the material filling them; if the septicæmia does not kill the animal at this stage, the next thing is necrosis of the pulmonary tissue, which may be entirely interlobular, or may complicate quite a number of lobuli; the character of the mass filling these spaces will be either sero-purulent, sero-puriform, or sero-hemorrhagic, with either of the other two conditions. In the center of such conditions may be often seen broncho-pneumonic centers, which, if freshly formed, have still retained their consistency. Did the destructive processes originate from the bronchial tubes, we should be able to find corroded and destroyed remnants of the same, and these conditions would bear relation to the dispersion of the tubes in their extent, which is never the case; their extent being invariably in correspondence to the parts shut out of circulation, which is invariably sharply outlined by the engorged vessels. Another thing which contradicts the broncho-pneumonic origin of these destructive processes is, that the anæmic, œdematous, purulent, or puriform conditions are fully developed, while those of the broncho-pneumonia are still too circumscribed in character to justify any such interpretation. Total, dry, yellowish-grey caseation of large sections of the lungs, or whole lobes, as occurs in phthisis in man, is never seen. The œdema is invariably limited to those portions of the lungs shut off from the circulation. Collateral œdema is never seen.

My esteemed colleague Dr. H. J. Detmers, who has made very ex-

* These processes in the lungs are surely of a hæmatogenetic nature, and seem to be the first that have ever been described of this character in any specific disease, for Baumgarten says (*Lehrbuch der Pathologischen Mykologie*): "As inciters of hæmatogenetic pneumonia the only known micro-organismal causes are staphylo and streptococcus pyogenesis." p. 270.

tensive and, in general, very trustworthy observations upon the swine plague, has come to some quite erroneous conclusions as to the genesis of the lung lesions, yet his testimony is valuable as showing that pneumonia is a common occurrence in this disease, even when the intestinal lesions are absent. He says:

“The most essential difference between the morbid features presented at the *post-mortem* examinations previous to December, and those found in the animals examined in the winter and spring, consists in a more frequent affection of the large intestines in the summer and fall, while in the winter and spring the principal seat of the lesions was almost invariably in the organs of the chest, but especially in the pulmonic tissue.”

Detmers then proceeds to give his conclusions as to the cause of the above variation in the organs complicated, which are as follows:

“This difference, it seems to me, is not accidental, but admits of an explanation. At any rate, the predominating affection of the organs of the chest, and especially the extensive embolism and exudation in the lungs, observed in every case in which the large intestines were free from ulcerous tumors, may be traced to distinct causes, acting principally during the winter.

“Swine, especially in the cold season of the year, on entering their lairs and going to sleep in the evening, are in the habit of crowding close together, or lying on the top of each other, and of frequently passing the night in very close quarters. Such crowding into a narrow space cannot fail to heat their bodies, to vitiate the atmosphere, and to accelerate respiration. Consequently, it will prevent a proper decarbonization of the blood, and retard its circulation in the pulmonary capillaries, and cause more or less congestion of the lungs, and prepare those organs for just such morbid changes as are effected by the bacilli or their germs. In the morning, after the animals have been heated during the night, and on arising from their lair in search of food, the air, especially in winter, is usually cold and chilly, and but a moment ago reeking and steaming with perspiration [Sic!—B.], they become chilled and commence to shiver. Such a sudden change of temperature necessarily causes a disturbance of the functions of the lungs and of the skin, contracts the expanded capillaries of the latter, and thereby compels the blood to rush to the heart and to the interior parts of the body. All this cannot fail to predispose, especially the lungs and heart, to become the principal seat of the morbid processes of swine plague, if the infectious principle (the bacilli) have entered the organism.” Ag. Dept. Report, 1879, p. 371.

The real cause of the predominance of pulmonary lesions in the winter and the intestinal in the summer and warm months, is not to

be sought, as Detmers concludes, in the "crowding together or lying on top of each other of the swine," but rather in the conditions of the surroundings. In fact, a better example that swine plague is an "extra-organismal" infection—in genesis—could not be asked for. In the warmer months (these conditions apply especially to the western states, where swine are not penned very closely in the summer,) the swine root and feed and wallow in the dirt of the infected localities, and the chances for intestinal infection predominate; they seldom seek their pens, or even are allowed to, but in the winter the ground is frozen hard, and rooting, etc., is impossible. During the warm months the germs in infected pens have been protected and multiplied in the straw and litter, which being protected from the rains is dry, so when the swine are penned, or seek these shelters in the winter, they find them full of a dry, dusty material, which is full of germs, and hence we have in such cases innumerable examples of aspiration broncho-pneumonia. But the broncho-pneumonia, only, has its origin in this way, as has been previously shown. This brings us to the discussion of another similar and equally misunderstood and complicated point in the pathology of this disease, viz.:

THE INTESTINAL LESIONS IN SWINE PLAGUE.

CAN SWINE PLAGUE BE INDUCED BY FEEDING?

In his summary of "the differences between the bacterium of hog cholera and the microbe" of swine plague, Mr. Salmon says of the—

"Hog Cholera Bacterium.

"Microbe of Swine Plague.

"Feeding cultures, after starving for a day, produce extensive necrosis of the mucous membrane of the large intestine; inflammation and occasional ulceration of the stomach and ileum." Report 1886, p. 675.

"Feeding cultures produce no effect whatever." l. c.

He repeats these assertions in the *Journal of Comparative Medicine*, l. c., p. 143.

Although one feeding experiment, the only one then made, in 1886 had given negative results, my extensive studies of this disease in the field had shown me that the second assertion was unequivocally false, and as to the first, that is of the same nature, as no such bacterium

exists in swine plague. I did not continue these experiments at the time, because I had no pens at my command which were of such a nature as to entirely eliminate the possibility of infection in any other way.

The proper conveniences being finally obtained, consisting of galvanized iron cages, with drawers of the same nature, which could be thoroughly washed and disinfected after each feeding, it was determined to proceed with feeding experiments in both rabbits and pigs. No preparatory treatment of the animals was introduced, as I believe that if a specific germ does ever cause infection by any special tract, that it must occur as under natural conditions, hence I emphatically condemn the value of results received by previous starvation, as Mr. Salmon claims to have done, or by neutralizing the chemical reaction of the stomach by alkaline solutions or by the injection of opium at the same time into the abdominal cavity, as Koch and others have done in Europe. All such treatment seems to negate the value of experimental evidence. Hence my animals were fed their regular meals at regular times. After eating, the utensils were removed and cages washed and disinfected so as to prevent any possibility of infection via the respiratory tract through the desiccation of the material. The results actually contradict Mr. Salmon's assertions and confirm our practical observations. They are also exceedingly instructive in other ways.

FEEDING EXPERIMENTS.

On the morning of June 5, 1888, I fed two one-third grown rabbits with some freshly pulled lettuce from my own garden. Before giving it to the rabbits a bouillon culture of the swine plague germ of known virulence was poured over the leaves of lettuce, and the superfluous quantity shaken off. The rabbits were placed in a large galvanized cage, which had been most carefully scrubbed with scalding hot water and then washed with 5 per cent carbolic acid solution. It was ten A.M. when the rabbits were fed. On looking at them in the afternoon the lettuce had been eaten up.

10 A.M., June 6. One rabbit just dead. Autopsy (No. 16) made immediately. (The other rabbit still apparently well; fed again in the same manner, and ate readily.)

The superficial subcutaneous veins were not nearly so much engorged as in cases of inoculation with a similar material. Abdominal cavity contained 10 grammes of a transparent fluid of a very delicate

straw color, in which the micro-organism was present, though not in great numbers. Blood of a dark, blue-red color, partially coagulated in the larger vessels. Contained but a few bacteria. Peritoneum clouded and swollen. Mesenteric vessels engorged, as well as those of the small intestine, which was also of a bright, diffuse, light pink-red color. Only the larger vessels of the cæcum and colon engorged. Those of stomach very much engorged. Lymph-glands swollen and of a diffuse, light red color. Spleen swollen, full of blood, in which many bacteria were present. Kidneys swollen, cortical substance slightly anæmic and clouded; medullary of a bright red color, except on limits of cortex, where a dark red line of engorged vessels manifested itself. Bacteria present. Liver swollen, very full of blood; acini clouded; gall-bladder very much distended; bacteria present in covering-glass specimens from liver. Stomach half distended with food; mucosa swollen, and covered with a viscid material, but of a diffuse pink-red color. Contents of small intestine semi-fluid, being mixed with an excessive amount of viscid, clear, slimy material; mucosa intensely swollen, and of a diffuse red color. The transverse folds in the large intestine were immensely swollen, in comparison to their condition in inoculation experiments, and of a diffuse red color; the contents were pultaceous and mixed with the same kind, though less in quantity, of pellucid, viscid material as seen in small intestine. Thoracic cavity: About a tea-spoonful of clear fluid in cavity. Bronchial and thymus glands swollen; diffuse red in color. Other organs normal.

June 6, 3 P.M. The other rabbit died. Autopsy exactly the same as the previous one, except that the catarrhal and hyperæmic conditions were more exaggerated.

From these two experiments it would seem as if the bacteria exerted their action in the small intestines by feeding experiments, or natural infection. Perhaps a less virulent material might show as much action in the large intestine in rabbits, but not having the rabbits to spare, that question cannot be tested at present. The bacteria were most plentifully represented in the second rabbit also.

FEEDING EXPERIMENTS BY SWINE.

On June 7, 1888, two three-months-old, black pigs were put into two large, galvanized iron cages that were perfectly clean, and in which

no experimental animals had ever been placed. No. 1 was then fed three times a day with 30^{ccm} of a bouillon culture of the swine plague germ herein described. This material came from the small pig that was brought from Mr. Burnham's, and was what constituted a weak case of natural infection.

No. 2 was fed with the same material used in feeding the two rabbits previously mentioned. In both cases the feed was meal and water.

June 8. No. 1 eating well.

June 8. No. 2 somewhat off feed.

June 9. Both eating fairly well.

June 10. Neither eating so well as the day before, and inclined to bury themselves in the straw.

June 11. No. 1 eating about as yesterday.

June 11. No. 2 not eating, but drinking frequently in the morning; buried in straw balance of the time. 3 P.M., dying; took temperature, as the animal was excessively hot to the touch, 106.5° F. Died shortly after.

AUTOPSY XXVIII.

Fairly good condition. No discoloration of cutis. On cutting open the skin and removing fore limbs, blood from axillary vessels very dark blue in color, and very thick. Exterior inguinal, axillary, and glands of throat excessively swollen, and juicy.

Abdominal cavity contained 250 grammes of a clear exudate. Peritoneum of abdominal walls swollen and clouded, a few petechiæ in substance. That covering the small intestines of an intense, diffuse, bright pink color, interrupted by the engorged conditions of the larger vessels, which were dark, blue-red in color. Same of large intestines, except that the diffuse pink color was in patches and not over the entire gut. Mesenterial vessels excessively engorged; the mesenterial glands were swollen as I have never seen them in any case, forming a packet over an inch thick and about three long; hemorrhagic centers were very rare. Serosa of the stomach marked by numerous hemorrhagic centers of a diffuse character and innumerable petechiæ.

Liver intensely swollen, edges very round; outside surface of a general pale red color, diversified by innumerable centers of a

yellowish-red color and varying dimensions; cut surface anæmic, of the same character. Acini distended; no vessels visible; very fatty.

Spleen swollen; contents blue-red and juicy.

Kidneys swollen; capsule non-adherent; outside and cut surface of cortex absolute anæmic, very much swollen, opaque, of a putty color; medullary injected.

Stomach half distended with ingesta. Mucosa excessively swollen, and in base gathered in deep rugæ about half an inch high; the same was covered with a viscid material.

Intestines: The mucosa of the entire intestinal tract was intensely swollen, over half an inch thick, and gathered in folds, which gave to the outside of these organs an appearance which I can only compare to a laminated gun barrel, except in color; there had been an active proliferation of the cells in the crypts and follicles, which was in reality a cause of the swelling, which was increased by considerable œdema. The contents of both guts were fluid, but more so in the small than the large intestine. No circumscribed neoplasms in the cæcum or colon; no follicular ulceration in either, but in the rectum there were numerous caseous centers of about the size of half a small pea, which extended above the orifice of a follicle, from which also could be pressed the same kind of a material; the follicles were manifest by their marked swollen condition, so that their orifices extended above the balance of the swollen mucosa. (See Plate XI., which illustrates a case of a similar kind under natural infection.)

Thoracic cavity: About a tea-cup of a clear effusion in same. Pleuræ swollen and clouded. Pericardium same. Contained a tablespoon of similar effusion. Heart muscle anæmic, yellow-grey-red in color, and very friable. The lungs were marked by innumerable sub-pleural petechiæ, which were also scattered through the substance. Anterior and middle lobes hyperæmic, atelectatic, and œdematous; diffuse in character. Tracheal and bronchial mucosa swollen and injected. An examination of the blood and organs, covering-glass, gave bacteria, but not so many as I expected to find. Cultures developed in tubes sown from the liver.

This case is very instructive to my mind. The very small number of bacteria in covering-glass specimens prepared from the blood and organs seemed at first rather surprising* when the severity of the disease and the strikingly dark color and thick consistency of the blood is

taken into consideration, but when one reflects more intently upon the lesions presented, the real value of this case becomes more and more apparent, and is such conclusive evidence to the disease being primarily a septicæmia that nothing more can be needed. But it is even more than that in a very essential sense! It positively shows that these bacteria, at least, can produce all the lesions of septicæmia without the necessity of their being themselves in the blood, and there producing this lesion by their direct bio-physiological processes and action upon that fluid.

In this case these processes took place within the intestines, and as much if not more within the small as the large intestines as in the rabbits. As noticed, the entire mucosa was in the same swollen condition throughout. This condition was not due to mechanical irritation of the germs, as the surface of the mucosa was absolutely intact, except the few small follicular lesions in the rectum. On the other hand, the entire glandular structure of the intestinal wall was more severely complicated than I have ever seen it; but had the septicæmia not been so excessive, and the blood more replete in micro-organisms, we should then have found these conditions complicated by the excessive hemorrhagic conditions of the lymph-glands and membranes, seen in acute cases under natural infection.

In this case, again, an exactly similar condition to that seen in the lymphatic structures of the intestinal wall extended to all the glandular structures of the same kind, but nowhere so severely as those in direct connection with the former, viz.: the glands of the mesentery. These conditions, then, point with absolute certainty to the development of the toxin which, being in solution in the intestinal canal, caused an hyperæmic condition of the same with serous effusion into the canal, and the very fluid-like condition of the contents; that this substance, or substances, exerted then a chemical, irritative action on the glandular elements is beyond question, for each gland, or better, its epithelium, was in a condition of active proliferation. This material then followed the usual course of the chyle ducts, or lymphatics, through the mesenteric glands, where the same kind of a parenchymatous irritation was set up; from here it passed into the circulation in the usual way, and, as said, septicæmia was produced almost, but not entirely, free from any influence of the germs in the blood, as there certainly were not enough present to cause the

strikingly pathological condition of that fluid. From here, the parenchymatous changes of the liver, kidneys, etc., were produced by the action of this same chemical irritant upon the caloric centers and the circulation.

Taking these strikingly peculiar and seldom conditions into careful consideration, it does seem as if no more valuable or conclusive experimental evidence could possibly be produced.

FIG NUMBER ONE.

It will be remembered that we left this animal comparatively well in appearance on the 11th, and that it was fed with a culture of much less active virulence than the previously mentioned animal. On the morning of the 12th of June, No. 1 ate its breakfast fairly well, but had a temperature of 40.50° C. On examining the body numerous petechial and ecchymotic spots were to be seen in the thin skin of the inside of the fore-arms and thighs and under the neck. At 4 P.M., when I examined it again, a greenish-yellow, aqueous discharge was being frequently passed from the rectum.

13th. Not eating at all. Temperature 42.10° C. The spots previously mentioned had become much more intense and diffuse, but no swelling was present in the parts; diarrhoea continuing.

14th, 10 A.M. Temperature 38.50° C. Animal prostrate; breathing labored, rattle in throat; a very fluid discharge of the same greenish-yellow color came away from the rectum at frequent intervals. 11 A.M. animal dead.

AUTOPSY XXIX.

The condition of the skin having been previously described, there is no need of mentioning it again. Blood purple-red in color and quite thick. External lymph-glands swollen and marked by a few petechial hemorrhagic centers. Abdomen contained about a tea-cupful of a clear fluid. Peritoneum swollen. Small intestine: vessels engorged, and serosa of an extremely delicate pink color, which became darker in the ileum, and still more marked in the large intestine, where the vessels were also more deeply injected. These conditions were still more marked in the serosa of the stomach. Mesenteric vessels as engorged as they could possibly be, and lymph-glands excessively swollen; a few petechial hemorrhages present.

Liver swollen. Outside surface mottled, the ground color being

reddish-brown, while numerous reddish-yellow centers disturbed the monotony. Cut surface more bloody than in previous case; acini swollen, and either cloudy or of a yellowish-red color.

Spleen enlarged, cut surface purple-red, and very juicy.

Kidneys swollen. Outside and cut surface of cortex swollen, of a reddish-yellow color. Some few vessels to be seen here and there. Medullary substance injected.

Stomach about one-third full. Mucosa swollen, deep red in color, with occasional hemorrhagic centers, and covered with a heavy viscid coating. Duodenum and jejunum: mucosa swollen, but anæmic, contents semi-fluid, and very viscid. Ileum: mucosa light pink in anterior parts, but of a dark, diffuse red color, growing darker as the cæcum was approached; contents same, but contained more solid material. Cæcum, colon, and rectum: mucosa swollen, of a pink-red color, interrupted by diffuse patches of a very dark red color. The walls of the cul-de-sac were thickened, the mucosa very rough and swollen, and covered with a diffuse mass of a yellowish-grey color, which broke up into flakes of a very friable character when removed; the tissues underneath were reddened and presented a granulous surface. The previously mentioned dark red patches were in exactly similar condition; they became less frequent in the rectum. No indications of the circumscribed neoplasms.

Thoracic Cavity: About a table-spoonful of a clouded fluid present. Lymph-glands swollen. The surface of the posterior lobes of the lungs was of a dark red color, and resistant to the touch; on cutting open, these parts were œdematous, the lobuli were of various shades of red, atelectatic, some consolidated, interlobular vessels injected and filled with a dark red coagulated material. Anterior and inferior portions of middle lobes consolidated; some of the lobuli were of a very dark diffuse red color; others were grayish-red, while still others were anæmic and yellowish-red in color; the interlobular vessels were as above. These parts were also œdematous. Tracheal and bronchial mucosa swollen; vessels injected; a yellowish-red, frothy fluid filled the lower parts of the trachea and larger tubes, while in the smaller fluid of the same color was present. No evidence of broncho-pneumonia. The blood of this animal, as well as the organs, also contained comparatively few bacteria, in comparison to the number found in cases of natural infection, especially where the germs gain entrance.

via the respiratory tract. In fact, the more one has experience in this disease, the more does it become evident that infection in this way is by far the most common, and that that by the digestive tract only is a rather rare occurrence. These experiments positively show that the so-called "characteristic lesions" in the large intestine in swine plague are of embolic origin.

GENESIS OF THE INTESTINAL LESIONS.

Here again we have three genetic forces to consider, and shall find the conditions described for the lungs almost exactly repeated.

1st, As in the broncho-pneumonia, the direct irritative action of the germs is in the intestinal tract.

2d, Their action through their toxin and the effects of fever upon the parenchymatous organs and the circulation.

3d, Their mechanical action.

The action of the germs as a direct irritant upon the mucosa of the large intestine is as specific as the genesis of the broncho-pneumonia. It is decidedly ulcerative or mildly irritative in its character, starting in the follicles and extending irregularly from them. We must shortly distinguish between a mild and an acute irritation if we will understand these conditions. The former is characterized by the production of a caseous coating, which takes its start in the orifice and canal of the follicle and extends to the tissues around it, the underlying tissues being but slightly congested, and is due to mild irritation of the epithelium and induration of the tissues around the follicle. Plate XI. The acute irritation, on the contrary, is marked by circumscribed interference with the circulation around the follicle and more ulcerative destruction of the mucosa. When this disturbance is very severe these lesions have a diphtheritic character. The diffuse diphtheritis on the other hand, which one finds only in the most severe and acute cases, is due to extreme disturbances of the circulation throughout the entire extent of the intestines by which a condition of general hyperæmia, approaching stagnation of the circulation, is present. Even the latter sometimes occurs, and then we find great patches of the mucosa in a necrotic condition and sometimes partially exfoliated. In such cases of natural infection, the respiratory tract must have given the atrium and great numbers of very virulent germs taken in at one time.

These conditions are to be most decidedly differentiated from the so-called "characteristic" intestinal lesions in swine plague. These are due to capillary embolism, and are sure evidence that the circulation in the large intestines of swine is of the so-called terminal variety, as in the lungs and kidneys. That is, the arterioles lose themselves in their peculiar capillary *rété* without any anastomoses, and this directly extends into a venous network, which unites in a small vein and then extends to the larger. The causes of this embolism have already been given in speaking of the conditions of the lungs, and the very reason these lesions are not so frequent as those in the lungs in swine plague is to be sought in the fact that the conditions favoring such embolism are not quite so pregnant as in the lungs; that is, the conditions to a continuous circulation of the blood are better in the intestines than in certain parts of the lungs. These so-called "characteristic lesions" may also be ulcerative-destructive, and neoplastic. The latter are by far the most frequent! In fact, in over 500 autopsies I have only seen one well-marked case of the former. The ulcerative-destructive disturbances of this character depend entirely upon the almost immediate embolic obstruction of the circulation, thus at once depriving a certain section of the intestinal wall of nourishment. In such cases we have intense venous engorgement, intense swelling, necrosis, and the formation of a circumscribed diphtheritis, and an exceedingly ulcerated condition of the underlying tissues. The edges of such an ulceration will be very ragged and swollen, and differentiated from the adjacent tissue by a furrow of more or less distinct character. Outside of this umbus the tissues may be, and frequently are, entirely normal. Plate IX.

When the embolism is formed more slowly, we find a circumscribed proliferation of the epithelium beginning within the follicle and extending outward in a radial manner, like so many rings around a center which is somewhat more elevated; the underlying tissues are indurated. Inflammation, in an active sense, is entirely absent in the formation of these neoplasms. There is no rupture of the blood-vessels or diapedesis. Their extent corresponds exactly to the territory shut out of the circulation. Sometimes clusters of them may be seen, but each will have its distinct line of demarcation. They extend somewhat above the level of the intestinal mucosa. The induration extends to the serosa. Plates V. and X. There is still a

third or intermediate variety, in which the circulation is not shut off so abruptly as in the former, but not so slowly as in the previous form. Here we have time for vascular disturbances and considerable hemorrhage. The indurated tissues are covered with a caseous material, the surface of which is of a dark color, and the radial character of its production is not so distinctly marked. This caseous mass is much more dense than in the former variety (where in many instances it is entirely wanting)—see Plates VII. and VIII., and is frequently desquamated as a sort of eschar, but the underlying tissues are not ulcerated. Such masses are of a dull, coal-black color, but owe this to pre-existence of hemorrhage or diapadesis and not to coal dust, as Prof. Walley assumes.

In the most acute cases diphtheritis of the entire intestinal tract is not uncommon, but that needs no further discussion here, nor do the parenchymatous changes in the liver and spleen. The kidneys, however, demand some slight consideration. Before I published my anticipatory reports, all American observers had persisted in describing the kidneys as “normal,” or but slightly affected. Since then Mr. Salmon has learned how to describe the external appearance somewhat, as well as to give better descriptions of the intestinal lesions than he ever did before. The lesions of the kidneys are those known as acute, parenchymatous nephritis, and are generally present in a most severe form, except in those very violent cases that end in 24 to 36 hours, when hyperæmia and clouded swellings, only, may be seen. These lesions have been so completely described in my autopsies that I need not discuss them here, as every competent reader will understand them fully. On the other hand, I wish to refer to a peculiar lesion which is sometimes met with in swine plague, and which has been erroneously interpreted by every observer. This lesion is of embolic origin, but due to a micro-organism that is an accidental presence, and has nothing to do with the swine plague. Walley and other observers speak of the surface of the kidneys being covered with small, red spots of about the size of a pin’s head. Walley says they are engorgement of the malpighian tufts, or hemorrhages, within the capsule; at all events, his language is to be so interpreted that we must assume that he looked upon these red spots as intracapsular. The fact is, they are not. They are all and every one irregular in shape and diffuse in termination. They are all due to rupture of the blood-vessels! They are all extra-vas!

The ruptured point of the blood-vessels can be easily seen on microscopic examination. They lie in the outside limits of the cortical substance, and immediately strike the eye on removing the capsule, and are also distributed all through that substance. A study of the cut surface of such a kidney will at once convince any one that they are not intracapsular, but extravasations, as their irregular form and extent cannot then be overlooked. The singular thing about them is this: they are due to capillary embolism, but the cause of the same is an immense round-ended, spore-bearing bacillus, larger than *B. anthracis*; and another singular thing is, that they are never seen in acute cases and only in protracted ones.

I think these bacilli are the ones met with by Klein, as he says an object like *B. subtilis* is the cause of swine plague. I have named the organism the Reeves bacillus, to honor my friend, Dr. James E. Reeves, of Chattanooga, Tenn., one of our most accomplished microscopists, who first clearly demonstrated them to me in the tissues. I had seen them in hæmatoxylin specimens, but very indistinctly, and did not understand their connection with the lesions mentioned, but being pressed with work, I took advantage of Dr. Reeves' skill, who kindly made me a set of sections of the lesions in the organs, pieces of which I sent him. Dr. Reeves positively demonstrated the fact that only when these small hemorrhages were present were these gigantic bacilli to be found, and I have since confirmed his observations by the examination of a large number of sections from kidneys in which these lesions were absent. When present in the kidneys, Dr. Reeves also found a few individuals were to be seen in sections of the lungs of the same individuals. I am responsible for the connection of the tissues, and I have confirmed this observation also. I have also found them in sections of the liver of the same individuals. An examination of the capillaries of the walls of the intestines would be valuable in such cases, but the time at my disposal has been so very limited that an extended study of the tissues so far as the distribution of the bacteria is concerned, has been out of the question.

WHAT THEN IS THE SWINE PLAGUE?

It would seem, from the evidence furnished, that every competent reader must have become convinced:

1. *That there is but one swine plague in the United States known*

at present, and that all the descriptions heretofore given in this report, including Mr. Salmon's, only apply to this one disease.

2. That this swine plague is caused by the ovoid, belted germ herein described. Discovered by Detmers in 1878; rediscovered by me in 1886.

3. That the disease is not only a septicæmia, but a septicæmia of extra-organismal origin.

4. That it is not a contagious disease.

5. That its primary and specific lesions are those common to all septicæmice, viz., degeneration, discoloration or thickening of the blood, swelling of the lymph-glands, and more or less tendency to extravasations of blood, with the described changes in the dense glandular organs of the body and the heart.

6. That the pulmonary lesions, except the broncho-pneumonia, are of a secondary character—the sequelæ of the septicæmia—and that when the former are present they simply indicate that the inficiens entered the body via the respiratory tract and that the septicæmia is still the specific lesion.

7. That the, so-called, “characteristic” neoplasmata (tumefactions) in the intestines are of a secondary origin, while the ulcerative and circumscribed follicular indurations in the large intestine, although indicating that the inficiens may have gained access by the digestive tract, still are no more primary lesions than those of the septicæmia.

INTRA-VITAL PHENOMENA IN SWINE PLAGUE.

A careful study of literature of this porcine pest from the European authors, with the exception of the British, will certainly convince even the most casual reader that they know very little about it. So far as we have any means of judging, the investigations of Professor Schütz, of Berlin, were made upon the cadavers, or portions of the same, of swine that were sent to him at his laboratory, and never in the field. Such kind of research can certainly lead to the discovery of the micro-organismal cause of any disease, but in such a diversified disease as this is, cannot demonstrate either all the pathological lesions which occur in it, but far less make one acquainted with its true clinical character and manifold symptoms. That the Germans know nothing about the swine plague, may be seen by the following quotation from their very latest author, which has reference to the Schweineseuche, however:

“But little trustworthy is known with regard to the clinical phenomena of Schweineseuche, as the disease has always been described under the name of ‘Rothlauf’ (erysipelas). In general, it can be said that the essential symptoms consist in its being a very rapid disease, which frequently terminates fatally in the course of a few hours, that the skin is reddened and much swollen, and that it is accompanied by cough and difficulty in respiration.” *Lehrbuch d. speciellen Pathologie der Hausthiere*, Friedberger and Fröhner, Vol. 2. p. 415, 1887.

The only satisfactory method by which we can obtain any clear understanding of the clinical phenomena in swine plague is to discuss them in direct connection with the causes which lead to them. They vary exceedingly, not only in different outbreaks, but in the different animals of one and the same outbreak these differences are fully as great; in fact this variation has been the cause of more or less skepticism as to the disease being the same in different parts of the country, and even farmers have often thought that the disease of an ensuing was not the same as that of a past year, though occurring in one and the same pen. This variation is to be sought entirely in the

character of the *causa sufficiens*—the germs. It does not seem probable that it can at all depend upon any quantitative relation of these organisms to that infected by them, or better, to the number previously gaining access to the porcine body, because of their power of rapid proliferation within the same, but rather to the qualitative powers of these organisms to excrete or give off the disease-producing toxic material. Germs that are naturally weak in this regard cannot certainly produce as severe a form of disease as those which possess this toxic quality in its full virulence. Had the true nature of this porcine pest been earlier understood, and had even those who have investigated this disease in this country in the past, been able to appreciate this fact at its true value, there is no doubt that the many uncertainties which now prevail as to its nature and symptoms would have long since been dispelled. Any peculiar idiosyncrasy of any one breed of hogs seems to be out of the question in this country, though European observers seem to have come to some such conclusion; some of these affirming that the finer breeds are more susceptible than the coarser or native hogs. Our experience in this country directly contradicts any such conclusion. On the other hand, when we can once make up our minds that the swine plague is primarily an extra-organismal infectious septicaemia, and that only, and that all other clinical phenomena or pathological lesions are but secondary complications resulting on and from the same, there will be no difficulty in understanding this very interesting and somewhat complex porcine pest. Being an infectious blood poison, which finds its origin outside the porcine organism, it is evident that the germinal cause must first gain access to the same. At present all the best evidence goes to show that the chief atrium by which the *causa sufficiens* enters the hog is by the respiratory passages; the second via the digestive organs. It is self-evident that the infection of hogs can also take place by both of these tracts at the same time. On the other hand, there is no doubt that the bacteria can gain access to the intestinal canal from the circulation, as we find them there even when we induce the disease by subcutaneous inoculation, which is far more conclusive evidence than an aspiration experiment. In such cases we shall find the same follicular disturbances as if the bacteria had gained access with food, etc. Third, via trauma, or wounds, which, however, are of very little importance. I do not know of a single case in which the evidence of

such infection has been present in any one of the numerous cases which I have examined. The fact that we can induce the disease by subcutaneous inoculation, though not invariably, seems to show that traumatic infection via the cutis can occur, but these very experiments demonstrate it must be the least frequent manner, especially as the chances of hogs becoming wounded, here in the West, where the disease most extensively prevails, is very small, when we take into consideration the absence of stone and other sharp materials in their pens, and especially in the large hog-runs.

In order to understand the clinical phenomena in swine plague, it becomes necessary then for us to take into consideration the action of the causal micro-organisms in the porcine organism.

THE ACTION OF THE BACTERIA IN SWINE PLAGUE.

It has been repeatedly stated that swine plague is an "infectious septicaemia,"—a blood poison—due to an, at present, unknown substance, or quantity, a ptomaine or toxin, which is produced by the bacteria during their development and given off by them to the blood in which they live; here it is held in solution and distributed over the entire organism. In our especial treatment of the morpho-biological characteristics of this germ it has been said, that we think that the white, or uncolorable, portion of their bodies represents this toxic, or poisonous part, and that it escapes from the capsule of the organism with the final separation of both of its terminal, or pole, ends. This toxin, or poisonous principle, is the primary and sole cause of swine plague. The bacteria of themselves do not cause the disease. Take away from them their power of producing this unknown element, which we can do, and which they will invariably lose of themselves in a longer or shorter time when cultivated artificially, and we can inject them into the healthy hog in immense quantities with no evil result. They will never regain this toxic power in the porcine organism. That they will in earthy materials, however, in a variable course of time, has been shown by practical experiences previously noted. It should be, therefore, evident that the primary clinical phenomena seen in this disease must be due to the action of this specific toxic principle. On the other hand, the bacteria exert another influence which must not be left out of consideration. It is their mechanical or obstructive action within the circulatory system. It is because of the utter inability of

other observers to properly value this mechanical or secondary action of the bacteria, that they have failed entirely in properly appreciating the true nature of the lesions of swine plague and their logical relation to the disease itself. It is also on account of their utter want of appreciation of the primary or toxie action of these bacteria that they have equally failed in determining the true pathological nature of the disease, both as regards its origin—contagious or infectious—and as to its character in the complicated organism. Hence, we find some laying great stress upon its being a pneumonia, Schütz, while others look upon it as “pneumo-enteritis” (Klein), while another endeavors vainly to make two distinct diseases out of one, Salmon. They one and all have overlooked the real disease. They have all endeavored to classify it by its secondary complications. They have all overlooked, or failed to appreciate, the essential lesions.

In order then to logically understand the clinical phenomena of swine plague, we must consider it from two points of view, according to the actions of the bacteria.

1. As a septicæmia.
2. As to secondary lesions due to this, and the mechanical action of the bacteria.

First, then, as an infectious blood poison.

Looked at from this point of view, the blood being saturated with a specific toxie material, of a chemical and irritative nature, which is distributed to every point of the body, we have not only to look for disturbances of a general character, but also for such in those organs or parts which are especially liable to irritation. While the material causing these primary disturbances is without doubt of specific nature, yet the earliest symptoms by which it manifests itself is absolutely without that character; it is a general disturbance due to the action of the toxin upon the heat regulating or calorific centers of the nervous system. Fever, or a rise in temperature, occurs, which varies in degree and the quantity of disturbance caused according to the virulent activity of the germs in secreting this specific irritating material; this rise in temperature may be from 104° F. to 111° F., as the two extreme limits observed in this disease. Much observation has taught me that this virulent or toxin producing activity of the micro-ctiological organism in swine plague, as well as the southern cattle plague, is almost if not entirely, dependent upon the time these germs have been developing

in the ground or some earthy material without having been frequently passed through several generations of animals and carried about from place to place. It also seems to me that the only influences having any pro or con action in this direction are excesses of moisture or dryness, and that excessive heat or cold have very little influence, as can be seen from the consideration of the very virulent outbreak which occurred at the college farm of the University during the months of December and January, 1887-88, during which we suffered from the notorious "blizzards" of that time, when the temperature fell to about 30° below zero for several days. As this part of the subject will be considered in a future report, it remains for me but to say, that where eruptions of swine plague occur yearly in one and the same pen or run, that the virulence of the disease does not seem to be as severe as when such outbreaks have only happened at intervals. This may be due to a certain degree of constancy of exposure on the part of the hogs, and a gradual infection per *vie naturee*; on the other hand, the numerical loss among the swine may not vary much each year, although the violence—acuteness—of the disease will be less severe than in outbreaks that occur at intervals, or new ones, in new localities, which result after hogs have been infected by diseased swine, in which the disease has been excessively violent and acute, and the germs have not lost their original virulence.

The first general phenomenon of constitutional disturbance due to the toxic principle secreted by the germs of this disease being fever, we must naturally expect the common result due to that condition, viz., general indisposition; very little desire to move about, burying themselves in straw or litter on the part of the diseased animal; shiverings; roughness and uncleanness of the coat; desire for seclusion; loss of appetite; thirst, and constipation. If the toxin production, on the part of the germs, be very excessive, the complicated animals may die during the early stage of the disease, no other phenomena than those accompanying the fever being present, as evinced in the case of experimental pig number 41 and the hog which died at the college farm on the 21st of December, 1887, full accounts of which have been given in the appropriate place.

In order to fully understand the clinical phenomena which may follow on the above, we must next consider the results of an excessive or prolonged rise of temperature in a diseased organism, especially

when, as in this case, the disease is of an acute infectious character. The first of these changes, in importance, is a greater degree of disturbance in the circulation of the blood, leading to hyperæmic conditions in various parts of the body, which are soon followed by ischæmic conditions in the great glandular and parenchymatous organs. The animal becoming weak and disinclined to move, the muscles do not require the normal supply of blood, and the intense fever causes a swelling of the individual cells of the muscle fibres, the parenchyma of which soon undergoes more or less fatty degeneration; this swollen condition of the muscles causes pressure upon the delicate capillaries running through them, thus shutting off the blood supply, and at the same time causing a reflux toward the larger veins, especially of the cutis and the internal parts, which gives rise to, or better, increases, the hyperæmic condition of those internal organs, the structure of which is such as to offer the least resistance to engorgement in their vascular system; of such organs the lungs are the most marked example. At the same time we find engorgement of the vessels of the dependent parts of the body—subabdominal region—with more or less obstruction in the vessels, which is followed by local hemorrhages and diffuse capillary discoloration of the cutis in such places. Similar changes to those in the muscles occur in the liver and kidneys. From a hyperæmic condition they soon pass into an ischæmic, their cells becoming swollen, and thereby causing pressure upon the capillary circulation, thus forcing the blood out of these organs by obstructing the arterial flow and pressing the blood out of the venous system, so that the already considerable plus of blood in the lungs and intestines, especially the veins, is still more augmented; such a condition must necessarily lead to inflammation if at all prolonged. The accumulation of the blood in those parts offering the least resistance to the same is, however, largely supported by another factor, and that is the condition of the myocardium (musculature of the heart), upon which depends the propelling power of the circulation. The heart being a dense muscular organ, much more so than any of the muscles of the body, it is but natural that its parenchyma should undergo the same changes as those already mentioned as occurring in the muscles and other parenchymatous organs. In no case are these changes more extreme than in acute, infectious diseases, such as the swine plague; in such cases we find the muscle of heart more or

less bloodless, the parenchyma swollen, yellowish-grey-red in color, and very friable. Such a heart cannot act; it cannot perform its work in normal, physiological manner. Hence it cannot drive the blood over the system as in health; hence a greater engorgement of the blood-vessels in the lungs and toward the intestines, and in the veins of the dependent portions of the body. The natural results following such conditions must be an irritable condition of the respiratory organs, leading to bronchitis and serous effusion in the smaller air tubes, and finally pneumonia. When the infection has occurred via the respiratory tract the symptoms of pulmonary complications will be among the earliest seen. Under such circumstances the clinical phenomena must of necessity consist of difficulty in respiration; coughing; still more excessive depression on account of congestive condition in the cerebral organs; a distressed appearance of the eyes; drooping ears; catarrhal outflow and accumulations in the canthi of the eyes and from the nose, with an increased scurfiness of the cutis. The hyperæmic condition in the lungs may assume such grave proportions, and the general disturbance be so severe, as to cause death at this time from want of sufficient oxygen to support the life of the animal.

We have, however, left the conditions in the intestines more or less out of consideration. The same hyperæmic conditions exist there; the peristaltic movements become retarded, the natural result of which is constipation; which conditions also tend to increase the hyperæmic condition in the lungs, which would be represented by still greater disturbance of the respiratory functions, which is again augmented by the fact that the active power of the heart has been continually growing less during the progress of the disease to this point. With the above description we have ended that of the specific clinical phenomena of swine plague, with two exceptions. Should we examine the urine of a diseased hog at this time, we should, in nearly every case, find that it contained albumen, and frequently casts such as are found in cases of acute parenchymatous nephritis, improperly called "acute Bright's disease." Such kidneys cannot act normally; they cannot perform their physiological functions; they cannot secrete urine as they should, and hence relieve the organism of many deleterious elements; the natural result must be the accumulation of such elements in the blood.

Among these are the so-called "urates;" hence, among the clinical phenomena of swine plague we must not be surprised to find those of uræmia, among which are cerebral depression or irritation, extreme weakness in movement, and above all, another phenomenon to which we shall allude in a moment. It has been said that the bacteria secrete a toxic, septicæmic, or blood poisoning material which is the essential or chief cause of all the previously described phenomena. When the kidneys cannot perform their physiological functions this material, and the urates previously mentioned, must naturally accumulate in the blood. It has been also mentioned that one of the points in which the blood accumulates in such diseases is the intestines. The urates and the specific toxin in such diseases are both of an irritating nature to the walls of the delicate capillaries which are of themselves distended to their utmost in such cases. Under such circumstances it is a well-known fact, which is frequently taken advantage of by physicians, that the intestines are either forced, or can be forced, to do vicarious work for the kidneys; these toxic elements thus irritating the walls of the capillaries of the intestines, together with the pressure upon their weakened walls, sooner or later leads to the passage of the fluid of the blood, carrying with it such elements as it holds in solution, into the intestinal canal, which thus changes the previous constipated condition into a catarrhal, and hence the choleroïd symptoms seen in swine plague, which have given to it the erroneous name of "hog cholera." Let me here again remark that the so-called "ulcerative" conditions frequently seen in swine plague have no connection whatever with these choleroïd phenomena, the causes of which have been given above, and which will appear sooner or later in the disease, in accordance with the time in which these combined toxic conditions of the blood and engorgement of the intestinal capillary circulation take place.

With these remarks we have concluded our description of the phenomenology of swine plague proper, that is, as an infectious septicæmia, with the exception that nasal hemorrhage sometimes occurs, and that the alvine discharges are of a dirty green color and very offensive odor when of a catarrhal nature; when the animals are constipated the feces are passed with more or less difficulty, and consist of hard balls of a blackish-green color. The above is a description of the clinical phenomena of acute swine plague in optima forma.

But this is not all !

The above described conditions and phenomena are all due to the specific action of the bacteria, their toxic action.

We have now to consider conditions and phenomena due to the secondary, mechanical, or obstructive action of the bacteria of this disease. The major clinical phenomena will not be changed thereby, but will be most decidedly increased in severity, in certain directions, more especially those connected with disturbances in the respiratory organs and the circulation.

We have previously considered the latter, but it is now necessary that we do the same with still further regard to detail. We have now to discuss the action of the germs of swine plague as bacteria only, without any connection with their specific or toxic action. In this case their action is mechanical only. It has been previously shown that changes had occurred in the organism which were most detrimental to any free circulation of blood over the diseased organism. It has also been said that the lungs, from their peculiar structure, offer the least resistant or supporting tissue to walls of the blood-vessels of any organ in the body; these vessels, therefore, become intensely engorged and distended with blood, especially in such portions of the lungs where the conditions to the natural circulation of the blood are the least favorable from their very position; non-movement of the blood in the blood-vessels is soon followed by more or less disintegration of their walls from want of nutrition, especially in the capillaries, which are only nourished in this way, having no nutrient vessels of their own; non-circulation of the blood in connection with diseased walls to the blood-vessels are the two essential factors to the coagulation of the same within the vessels during life. The presence of any foreign body within a blood-vessel is soon followed by the coagulation of the blood around the same. In swine plague we have just the conditions necessary to the coagulation of the blood. The blood is replete with bacteria, each one of which is a foreign body, the diseased wall of the vessels is another, and the stagnation of the circulation fills the essential bill to coagulation and the complete obstruction of the circulation in such parts; the more distant such parts are situated from the direct energy of the circulating stream, the more the heart is complicated, the greater the stagnation of the circulation, and the greater the tendency of the blood to coagulate in the finer blood-

vessels, which tendency is increased according to the number of the germs collected in such vessels; hence it is that certain parts of the lungs frequently become entirely shut off from the circulation, which must necessarily cause an increase of the hyperæmic condition of the more favorably situated parts, which condition manifests itself clinically by the augmented difficulties in breathing of the suffering animal, and increases the dangers of a fatal termination of the disease. To the same cause, capillary obstructions of the above mentioned character, is due the hemorrhagic condition, as well as local gangrenous centers, frequently seen in the skin in swine plague in more protracted cases. It is scarcely necessary to touch upon the results of the physical examination of the thoracic organs in cases where they exist in swine plague, as such examinations are seldom made, and it is evident to the competent examiner what such results must necessarily be under the circumstances herein described.

DIFFERENTIAL DIAGNOSIS.

WHAT CONSTITUTES IDENTITY IN GERM DISEASES?

MORPHO-BIOLOGICAL RESEMBLANCES OF MICRO-ETIOLOGICAL ORGANISMS NOT SUFFICIENT EVIDENCE UPON WHICH TO ASSERT THAT THE DISEASES FROM WHICH SUCH ORGANISMS ARE DERIVED ARE NECESSARILY ONE AND THE SAME DISEASE.

Though Professor Schütz fell into the error of considering the second series of diseases of which he treats, in his report, as identical with the first series, which were identical with the disease described by Loeffler, on account of the apparent corresponding resemblance in their micro-organisms, but, as has been shown, the lesions in the different animals do not correspond sufficiently to warrant any such conclusions, those of the second series corresponding to the swine plague proper, only wanting the intestinal lesions described by Roloff to complete the picture, the absence of these lesions in three swine, the number examined by Schutz, is not surprising, as I have met with a very large number of cases of swine plague where they were wanting.

On the other hand, Schütz was very well aware that an error could, or can, be made in diagnosis from giving too much value to morpho or cultivatio-biological resemblances in micro-organisms as is well shown in the following passage. He, himself, as has been previously noted, seems to have been fully aware of the fact that he had a very different set of lesions under observation in his series of swine—those from Pülitz—than he had in the first, for he expresses his astonishment at finding the germs so alike:

“Hierbei ergab sich zu meiner grossten Ueberraschung dass in den hepatisirten Theilen der Lunge grosse Mengen ovaler Bacterien von verschiedenner Grosse nachzuweisen sind.” 1. c., p. 382.

Finding the germs alike, however, could not free him from a certain deference to the authority of Loeffler, though in the next passage he says:

“When the bacteria discovered in the lungs (of the second series) correspond in their form with those found in spleen (first series) it is not to be at once determined that they are identical with the same. We also know that correspondence in manner of development is not sufficient grounds for us to assert that two such organisms are identically the same. As an example I will call to mind the fact that we are acquainted with a bacillus which morphologically cannot be distinguished from that of anthrax, and which also develops in the same manner in meat infusion, gelatine, and other media, and only through its physiological attributes distinguishes itself from bacillus anthracis, as it produces no injurious effects when inoculated upon animals.” l. c., p. 393.

Schütz also saw the real ens of patho-differential diagnosis when he afterwards uses language which amounts to the same thing as the Scriptural saying, “By their fruits shall ye know them,” and still, as I have endeavored to show, he did not sufficiently appreciate, or correctly taste of “the fruits of tree knowledge.” This never to be neglected principle, “by their fruits shall ye know them,” has been altogether too much overlooked by the so-called bacteriological school of investigators. It has been a psychical anomaly which has disturbed the mental accuracy of many investigators other than our Mr. Salmon, in whom it seems to have taken a very erratic and myopic form, that of seeing microbes which do not exist in swine plague and then describing them. The followers of this bacterial school seem to have been very short in a certain kind of mental food; their appetite for priority-notoriety seems to have been a sort of psychosis; they have suffered from an insatiable hunger to have their names connected with the discovery of some new germ, rather than to do really creditable pathological work.

The fruits are of more importance than the seed. Unless we know the nature of the fruits, the character of the seed, though important, is still of little account in preventive medicine. In this sense many members of the Koch school have fallen into the serious error of attributing caseation, and everything else, to the direct action of bacillus tuberculosis, whereas caseation is death to these bacilli; caseation being in itself death, it is evident that the bacilli cannot live in dead tissues. That caseation can and does occur entirely independent of bacillus tuberculosis cannot be better demonstrated than by studying the true swine plague. Schütz demonstrated this fact. Caseation

is dependent upon the character of the matrix ; the cells from which the neoplastic products develop and not upon the nature of the irritant which sets them into activity. The irritant may set dormant forces into action again; but the matrix will determine the character of the product. These observers have been altogether too much bacteriologists and not sufficiently pathologists for the best credit of the school out of which they sprung. People who knew nothing of pathology, or bacteriology either, so far as that goes, have said that Virchow is an active anti-bacteriologist. Quite the contrary is the case. Virchow is a pathologist and values bacteriology, in the only correct manner, by the effect of the germs in the infected organism. It is owing to the critical analysis to which the greatest of all pathological masters has subjected the work of the simon pure bacteriologists that a healthy reaction has begun, and the latter, like the master, are beginning to appreciate the fact that even bacteria must be judged by their fruits, more than any other way, and that it is not sufficient to gain a name as an investigator to discover a new germ, far more to manufacture one, and then describe that which never existed in the connection asserted.

We have under consideration a certain group of germs to which I have given the name of the "ovoid, belted group," to distinguish them from the others which have apparently the same action—that is, produce septicæmia. To this group belong the micro-etiological organisms of the rabbit septicæmia, the hen cholera, the wild-seuche, swine plague, the southern cattle-plague, and as I have shown, in all probability, the yellow fever and some other diseases, an example of which exists in Nebraska, though I have not sufficiently studied that point as yet.

Now these micro-organisms, so far as we know, all have much in common. They look so much alike that it is almost impossible to distinguish one from the other by microscopical examination. They nearly all react alike towards the different tinctions, though sufficient experience in this line has not been had to assert that there may not yet be discovered some points of differentiating value from this source. They also grow very much alike upon or in different media. Some say they have the power of idiosyncratic motion, while others deny it, but that seems to depend upon what the different observers consider motion to be. Mr. Salmon sees motion in one germ

at one time and immediately after says it has none. Loeffler says the species seen by him does not grow on potatoes, while Kitt says it does, and Hucppe says the same of the germ of "Wild-seuche," which is unquestionably the disease Loeffler studied. All observers agree that the germ of swine plague does grow on potatoes, except Mr. Salmon, who is myopic now and again, at one time saying it does and telling how it differs from his "hog cholera microbe" in this direction, and at another telling us it does not grow on these tubers. As I have pointed out in my report upon the southern cattle plague, this development of the members of this group of germs upon potatoes may yet serve as a means of differentiating them; for instance, upon this medium swine plague, when fresh, produces a colony of a peculiar greyish color (muddy coffee brown); that of the southern cattle plague, a yellow color; while the cattle disease germ referred to grows almost white. If we transfer these germs to cooked and sterilized white of eggs in the moist chamber, the swine plague germ grows as a whitish, semi-fluid mass, difficult to be seen by direct observation; the southern cattle plague colony becomes light buff, while the other one, instead of being white as before, is now a bright yellow.

Again, by their "fruits shall ye know them!"

The cattle plague germ produces that disease and does not extend to hogs or hens when exposed in a natural manner. (What can be done by artificial inoculation has nothing to do with the question.)

The swine plague germ produces swine plague and never extends to cattle, horses, or fowls, no matter how exposed, and is also noted for its special fruits, the characteristic lesions in the intestines and the pneumonia. Hen cholera is limited to the feathered tribe, but how many different species it extends to naturally I do not know. Rabbit septicaemia is an artificial disease, produced by a germ accidentally found by Gaffky and inoculated upon rabbits. I do not know of a well authenticated case of natural infection, notwithstanding the one reported from that source of bacteriological wonders, the bureau of animal industry at Washington.

"Wild-seuche" is another disease which is peculiar in its fruits; it comes naturally to pass in deer, cattle, and swine, and according to some, in horses. It is characterized by "enormous œdema" and an excessive tendency to hemorrhage, a tendency which is common to all

this group, but in a varying degree, and in none so severe and constant as in the "Wild-seuche." These diseases then have all several phenomena in common.

First—Their etiological micro-organisms resemble one another very closely morphologically, and they have striking resemblances when developing in or upon artificial media.

They are all blood poison diseases—septicæmiæ.

Third—Their germs all find the locus of primary development outside of the animal organism, hence they are extra-organismal septicæmiæ.

Fourth—They are all accompanied by more or less hemorrhage, hence the name "septicæmiæ hemorrhagica," as given by Hueppe, would not be a bad suggestion were it not that other septicæmiæ caused by an entirely different class of germs are also more or less hemorrhagic, anthrax, emphysema infectiosum (black leg), etc.

Hueppe, who is one of the most expert and competent bacteriologists living, certainly gave cause to a wrong impression when he classed several of the diseases mentioned as one and the same (which principle would equally well apply to the true swine plague, the southern cattle plague, and yellow fever), viz., rabbit septicæmia, hen cholera, the "Wild-seuche," and German "Schweineseuche." I desire here, however, to most earnestly apologize to him for criticising his assertion as to the "Schweineseuche," for I now fully agree with him that the Loeffler "Schweineseuche" (and the first series of Schütz hogs) is really the Wild-seuche, but I was misled by the "fruits" in Schütz's second series and his admission that Roloff's intestinal lesions also should probably be included under the "Schweineseuche." On the other hand I feel I was unquestionably correct in opposing the tendency of Hueppe's attempt at such generalization, for the "fruits" emphatically show that the true swine plague has no connection with the "Wild-seuche," Kaninchen-septicæmia, or the "Huhner-cholera," though the germs unquestionably belong to the same group, any more than the last three diseases have any necessary etiological connection with one another. However, I believe in letting every man speak for himself, and so will quote Hueppe to some extent and give his reasons in my own language.

Hueppe says :

"Even though I admit that the evidence is not complete, still, so far as my conclusions can be based upon biological studies, I must confess that the 'Wild-seuche,' 'Schweinesenche,' and probably 'rabbit-septicæmia' and 'hen cholera,' are only different appearing forms of one and the same infectious disease—the 'Wild-seuche, or septiciæmia hemorrhagica,' as he technically calls it. l. c., p. 788.

In a letter dated May 18, 1888, Hueppe writes me that I have mistaken his meaning, and that he had no intention of making this generalization apply to all diseases caused by members of this ovoid, belted group of organisms. Allowing this correction most cheerfully, I will let my manuscript stand as written, in order to prevent others falling into the same tendency, because I think it will sooner lead to the settling of the question in point as to the swine plagues.

That is just the trouble! Hueppe neglected to consider the fruits, and based his conclusion on biological studies, though he was, also, undoubtedly influenced by the two circumstances previously mentioned. The diseases he thus attempted to group together were not only all septicæmic, but at the same time more or less hemorrhagic. It was the clinical fruits he most seriously neglected. He seems to have appreciated the results of Roloff's examinations of swine in 1875, but did not see that they had relation to Schütz's second series of investigations, and not the first, nor did he see that the fruits, necroscopical lesions, in Schütz's second series did not correspond with those of his first, or with those of Loeffler. That he failed to see that the very brief clinical history of "Herr College Hirschel," insufficient as it was, in no way corresponded with that of the "Wild-seuche" is not to be wondered at in a purely medical investigator without much personal experience in animal diseases, but in this he is far more excusable than Prof. Schütz, who, with both at his command, should have valued his "Ueberrashung" and as a pathologist paid more attention to the fruits.

* Following out Hueppe's line of argument, and admitting that the "Wild-seuche" attacks deer, cattle, and swine, and that it can and does occur in one or the other species and not necessarily in the other; admitting also that it manifests itself by its patho-anatomical fruits, in pectoral and exanthematic forms which are generally complicated by lesions of the intestines; admitting that it is unquestionably an extra-organismal septicæmia, and that hemorrhages are fre-

quent, still that does not make it out that it is an identical disease with the rabbit septicaemia, or hen cholera, and far less with the swine plague, which, as we well know here in America, never extends to cattle or fowls, for were Hueppe here in Nebraska he could see a large drove of hogs feeding among cattle, the hogs sick with swine plague; many of them soiling the common food of both cattle and hogs with their alvine discharges; all of them lying among the corn husks and hay; the cattle continually eating fodder and corn soiled by the diseased pigs, and kicking up a dust full of desiccated germs, and all the animals drinking polluted water. I can assure him that he can see such cases in which 500 cattle and as many hogs are in the same field and that not an ox or steer will be sick though the hogs may be dying off at the rate of thirty or forty a day. He can also see hens, ducks, geese, and turkeys eating the same food and picking over the recently fallen faeces from the diseased swine; he can see these fowl eating the bodies of the diseased swine, but he would never see one die from the swine plague or hen cholera on that account.

Again, though essentially a septicaemia of extra-organismal origin, as is also the "Wild-seuche," Hueppe would find that in the swine plague the "enormous oedema" was invariably wanting, and the hemorrhages were generally, though not always, not so excessive, and above all, he would find the pulmonary lesions tending to a caseous or gangrenous character and less hemorrhagic, and what is still more important, he would find the Roloff intestinal lesions a very frequent, and when present, characteristic lesion. But what is of equal importance, he would find a disease the clinical course of which generally extends from ten to twenty days under natural infection, though he would also find cases of from twenty-four to forty-eight hours' duration, in which the hemorrhagic septicaemia would equal anything that probably is ever seen in the "Wild-seuche," and in such cases he would not find either developed pneumonia in any degree, or the intimation of the Roloff intestinal lesions, but he would find diphtheria of the intestinal tract, which, like old John Harper's horses of Kentucky, ran "from end to end of the track." Again, the diarrhoea which occurs in so many cases of swine plague is dirty green in color, not accompanied by tenesmus, being frequently the first symptom observed by farmers. It is very seldom accompanied by hemorrhage, while in an equal number of cases the most aggravated constipation is

present, the faeces being passed as hard, greenish-black balls; in such cases they may be often seen streaked with blood. Hence no matter how these germs may resemble each other when artificially cultivated or examined microscopically, they fail in the one great factor necessary to make the diseases produced by them identical. They do not have the same physiological-chemical attribute with regard to a given something produced; which invariably decides the primary pathological results produced by a given germ. Notwithstanding the latter fact, these diseases all have a very close relation to one another. They are all, as has been said, extra-organismal, local septicæmia in their primary origin. Each one, however, has something peculiar about it that prevents them from being identical diseases from any action of the germ.

Each species of animals in which they occur has some unknown constitutional idiosyncrasy which renders its members susceptible to the action of a given germ, and each of these germs has some peculiar unknown biological idiosyncrasy by which alone it infects, naturally, but a given species of animal life.

These two factors, together, can alone decide the identical question!

What we can do artificially, by the inoculation of the animals that the disease does not occur in naturally, has no necessary relation to the question whatever.

Notwithstanding the value of the peculiarities of micro-organisms in the general differentiation of many diseases from one another; notwithstanding the value of the "fruits" produced by such micro-organism in infected individuals, *still when it comes down to deciding between these septicæmiae caused by members of the ovoid, belted group of germs, I dogmatically assert that the clinical history, the limitation of the disease to one species of animals, or its extension to several, its duration and clinical symptoms are the points of differentiation upon which we can alone depend, and not upon the germ or pathological lesions.*

When we desire to produce the same disease by artificial inoculation we cannot be assured we have done so unless we produce the same disease in all its clinical history-phenomena, and pathological conditions that occur under natural infection.

PORCINE ERYSIPELAS.

*“ROTHLAUF” (GERMAN), “ROUGET” (FRENCH.)

As has been shown in another part of this report, this most interesting disease was looked upon as the swine plague of Europe until Loeffler and Schütz demonstrated that it was not, in the year 1885. The “Wild-seuche” had been known as an idiopathic malady since Bollinger’s investigation in 1878. Even in the work before me, the authors treat the Rothlauf as not only identical with the German “Schweineseuche,” but as the same disease as the swine fever of Great Britain and the swine plague of this country. It is therefore evident that, though Schütz made his investigations in 1885 which demonstrated a second pestiferous disease in swine in Germany, that his researches must have been made subsequent to the publication of this work by Lydtin and Schottelius. It is also evident that aside from the work of Klein in England, and Detmers in this country, nothing definite was known as to the micro-etiological causes of the different porcine pests of the world prior to the year 1885. As has been shown, while undoubtedly having seen the germ of swine plague as early as 1876, Mr. Klein gave such an erroneous description of it, by comparing it with *Bacillus subtilis*, that no credit can be given to him as a discoverer, and although Detmers also discovered, and quite correctly described, the germ of the true swine plague in 1880, still the credit of differentiating the Rothlauf as an independent disease belongs to Schütz, although Loeffler laid the foundation of the work.

As not a case of Rothlauf has yet been seen among the swine of the United States, it is comparatively safe to say that the disease does not exist here; at the same time it seems to me that this work would be, in a measure, incomplete, were not some of the essential characteristics of the porcine pest placed before the readers. Practical swine breeders will, it is to be hoped, not only see the differences between the swine plague and the Rothlauf, but will also see that the reason Europeans considered the porcine pests of their respective countries to be all one disease, was simply because swine died in numbers, but also because more or less redness of the skin existed in the majority of cases. In the Rothlauf, however, it is the essential intravital lesions, neither the

*Rothlauf der Schweine—Lydtin—Schottelius—1885.

pneumonia nor the characteristic greenish-grey discharges of the swine plague occurring in it. As I am opposed to making compilations from the work of authors in a report of this kind, I shall select such portions of the work of Lydtin and Schottelius as seem necessary, and offer very free translations of the same, taking the liberty of calling attention to any special points that may come to mind during the task.

CLINICAL PHENOMENA OF THE ROTHLAUF.

“The disease generally appears suddenly among a number of swine at the same time. [It seems that the disease is peculiar to swine, as we find no mention of it decimating other species of our domestic animals.] The sick animals are very apathic, and seek out of the way places, burying themselves in the straw or litter; their respirations are short and difficult, the animals groaning some and have little appetite or desire to drink. The voice of the patient is weak and hoarse, especially when the lymph-glands of the intermaxillary space and neck are much swollen. Constipation is marked, the fæces being covered with a slimy material, and often bloody. At the acme of the disease diarrhœa frequently sets in, the discharges being of a thickish, semi-fluid, blackish character. [The marked difference between these diarrhœa evacuations and those of swine plague will be at once evident to every one at all acquainted with the clinical phenomena of that disease.] The animals urinate but seldom, and in small quantities. The visible mucosa are either of bright or brown-red color. Temperature frequently ascends to 43° C. The animals at first lie with their limbs drawn up under the body, and are difficult to rouse up. Upon the delicate and bristle-free parts of the skin, such as the lower parts of the abdomen, the breast, and the neck, as well as the inner skin of the limbs, there appear numerous red spots in the course of the disease, which soon extend in a diffuse manner and occupy most of the skin, being at first red, then dark-red, violet, or even brown in color. This redness ascends from the inferior portions of the abdomen, up the sides, and over the backs of the diseased animals. Such places are no more painful or warmer than other parts of the skin that are not so affected. They are not elevated or swollen, and only distinguish themselves by their color from other portions of the body. [The difference between this disease and the “Wild seuche,” with its enormous, deforming œdema, should be at once apparent.] The afflicted animals gradually begin to breathe more rapidly and with augmented exertion, groan much, and become very weak and inattentive to disturbances. While at first weak, in general, they now manifest a decided weakness or even paralysis of the posterior parts, and under the phenomenon of general weakness, which is often

accompanied by convulsions, the animals finally die. The redness of the skin is not always apparent, especially in very acutely diseased animals, but then appears soon after death. Healthy swine which are placed in a pen with others having this disease, or in pens where such have been, but have not been cleansed and disinfected, show symptoms of infection seldom on the first day, but most frequently on the third and fourth and less often on subsequent days to the eighth. The duration of the disease varies sometimes being but a few hours before death results, while it may extend to four days. As a rule the fatal termination comes in thirty-six to sixty hours after the animals are first sick." [The difference between the duration of the Rothlauf and the swine plague is self-apparent when an average is taken, though, as is well known, cases of swine plague occur which terminate within twenty-four hours from the time the animals were first observed to be ill, but the average duration is certainly not under seven to ten days, and perhaps more often over than under that period.]

POST-MORTAL PHENOMENA IN ROTHLAUF.

"Rigor mortis sets in soon after death, but disappears again in rather a brief space of time. The body soon becomes distended by the generation of gases; when the body has been discolored, it soon assumes a bluish-black color, shading towards green, and the cadaver soon undergoes putrefaction. In animals that have been killed, or such as have just perished, the skin of the discolored parts is infiltrated with blood, œdematous, as well as the fat tissue underneath, to a considerable degree. The muscles of such parts are soft, slimy, pale red, and clouded in appearance, looking as if they had been soaked in water for some time. [Not seen in swine plague]. The subcutaneous veins are engorged with blood, especially in the vicinity of the lymph-glands; the latter being frequently imbedded in a gelatinous, hemorrhagic mass; they are swollen and of a dark red color; the cortical substance of the glands is of a brownish-red color, and the cut surface marked by hemorrhagic centers, the parenchyma being soft and juicy; the glands of the inguinal region, the intermaxillary space, the neck, the thorax, and the mesenterium generally present these changes. [The same changes in the lymph-glands are also seen in swine plague but more especially in acute cases.] The peritoneum is either clouded or covered with reddish striæ, petechial, or ecchymotic hemorrhages. The abdominal cavity either contains none or a small amount of a clouded, reddish fluid. [In swine plague abdominal effusions of considerable extent are quite common; they may be either clear, clouded, straw-colored, or hemorrhagic, according to the severity of the case.] The external surface of the stomach seldom shows any lesions, but the mucosa of the larger curvature is often deeply red or of a dark-

brown color. As a rule the mucosa presents a considerable engorgement of the blood-vessel, and is considerably swollen. The serosa of the small intestines and the attached mesentery is generally of a diffuse, red color, or marked by hemorrhagic centers of various forms and extent; marked vascular engorgement is never absent. The muscularis and mucosa are generally swollen, and here and there separated by hemorrhagic extravasations; the mucosa is swollen and of a bright red color; the crests of the folds are often bare of epithelium, and hemorrhagic; capillaries engorged. Solitary follicles as well as Peyer's plaques are swollen, especially those of the posterior portion of ileum; ulcerations are sometimes present in the vicinity of the ileo-cæcal valve. Aside from a viscid covering to the mucosa, which is sometimes pigmented in places, the small intestine is frequently quite empty. When we view the large intestine externally, it shows but few if any changes, though petchiæ may sometimes be present. The contents are either solid or, when this is not the case, of a black, semi-pultaceous character resembling wheel-grease; the mucosa is swollen, and may be either diffusely reddened or marked by hemorrhagic centers of different shapes and extent. Lesions generally fail in the rectum. Liver generally swollen; color dirty brown with paler spots; cut surface generally paler than the external; fatty; a semi-fluid blood from the same. Spleen generally swollen somewhat, and of a dark brownish-red color; pulp has generally a normal consistency, but sometimes replete in blood; in others it is degenerated and semi-fluid and black in color. Kidneys frequently swollen. Effusions seldom in the thoracic or pericardial sacks [frequent in swine plague]; engorgement of the vessels are, however, sometimes present and other small, petechial, or striated hemorrhages in the membranes. Lungs contain air in all parts, full of blood and red." [Pneumonia frequent in swine plague.]

The Germans say the disease is contagious, but owing to the uncertainty with which this word is used, and taking other things into consideration, it would seem as if some more critical light should be shown on that point. As speaking against the contagious idea, according to the only logical definition of the word, is the following passage: "The Rothlauf is an enzoötic disease in some districts, where the land has a peculiar character." It again differs from the swine plague according to this description in that, "the disease is seldom seen in young pigs as well as old hogs, but most frequently attacks those from three to twelve months old."

As against the assertion that the "Rothlauf" is a contagious disease of swine, and in favor of my idea that it is an extra-organismal infection, is the following from the work quoted:

“The experiments have also shown that the disease can extend from inoculated animals to those not inoculated [*i. e.*, to healthy swine]. The most exact investigation of this phenomenon has, however, shown that the transmission could not be charged to mere cohabitation of the animals, but that the infected animals had either consumed the fæces of those which were ill, or that they had eaten of the carcasses of those that were dead, or mice that had done the same thing and died therefrom.”

In another place the same authors speak of healthy swine becoming infected

“In consequence of the carelessness of an attendant who allowed them access to a manure pile where the dung from the experiment station had been emptied; the healthy swine rolled in, and ate some of the material.” *l. c.*, p. 188.

These statements being correct, I have no hesitancy in pronouncing the Rothlauf an extra-organismal infection and not a contagion. This disease is caused by a very delicate bacillus, of which we give an illustration, Plate III., Fig. 6, which, by comparison, at once distinguishes it from the swine plague, were we unable to differentiate it in any other way.

THE DIAGNOSIS OF SWINE PLAGUE.

As neither the “Wild-seuche” or the “Rothlauf” are known to exist in this country, and as the former is essentially characterized by attacking cattle as well as swine, as well as the deforming tumefactions of the skin, while the latter is marked by the peculiar redness previously described, and both by an, in general, far more acute course, and as neither have the really “characteristic” diarrhœa of swine plague, which will certainly be seen in some individuals, though not constant, in most any outbreak, it can be said that when hogs are dying in considerable numbers in any part of this country, that when they either appear constipated or have a peculiar greenish-grey, watery discharge from the bowels; that when they hide themselves in the straw, or are finally very weak, especially in the posterior extremities; that when severe lung complications are often apparent, and the average course of such a disease is from seven to ten or more days, though sometimes as short as twenty-four hours, and again extending to twenty or thirty days, that that disease is the swine plague.

PROPHYLAXIS.

THE PREVENTION OF SWINE PLAGUE.

The prevention of diseases of any nature must be largely dependent upon what, in a general way, we speak of as hygienic measures. With regard to diseases of a contagious or infectious character, the results of practical observation, supported by experimental research, have entered the path, long since opened, of what is termed inoculative prophylaxis.

Neither of these methods, either singly or collectively, will ever be found infallible. Each must be the necessary correlative of the other. Both are dependent upon practical observation and scientific research for their development and gradual completion. The word "practical," as used above, is not to be taken in the commonly accepted sense, but rather in the empirical scientific, where the diseased individuals are, or have been, the object of observation and reflection by competent and thoughtful observers. The logical conclusions thus drawn have all the value of those resulting from direct experimentation.

Old as the world is, scientific preventive medicine is yet in its infancy. Paradoxical and irrational as it may appear, it is far more developed, far better studied, in relation to the diseases of animal than those of human life.

It would be unjust to the medical profession not to say that this peculiar condition of things is the natural result of the fact that animals can be used for experimentation. This fact, however, may be, and will be, found to indicate the way to the prevention of some human diseases. Actual scientific experimentation upon animal diseases of a devastating character has scarcely yet progressed far enough to permit of any absolutely trustworthy generalization of the results to human diseases. But, if the results of my studies upon the southern cattle plague and the yellow fever both become confirmed in all their chief essentials, if the inoculative treatment for the prevention of swine plague can be so perfected as to be practical and safe, then these three diseases being caused by a similar yet not identical micro-

organism, but still being septicæmiæ, must be equally preventable by inoculation, and the work can be inaugurated for the yellow fever in man with equal safety to the diseases of the same kind in animals. Although I have no trust whatever in Frière's bacteriological investigations, I can see no reason to be too skeptical as to the value of his inoculative treatment of the yellow fever. It is a historical fact, as has been shown, that Pasteur successfully developed his "vaccine contra rouget," as proved by hundreds of inoculations, even when he did not know what micro-organism caused that disease; and though Mr. Salmon followed his misleading footsteps in regard to the "micrococcus," I am not sure but what the only correct assertion he has ever made about preventing swine plague by inoculation was when he said, of his experiences with that "micrococcus," "a reliable vaccine might easily be made if we carried our investigations but a little way farther." That is, if the true germ was in the material, and the experiments were genuine. Now as Pasteur was successful in rouget, because the specific "bacillus" was in his material, though he saw a coccus, so may Frière, following in the same direction, be successful in the yellow fever without ever having seen the germ, and Salmon may have had good grounds for saying what he did in 1883, though he never knew anything about the true etiological organism of swine plague until I demonstrated it to the world in 1886.

It has been said that this scientific side of preventive medicine has become better developed in relation to animal than human diseases. Here, in the United States, we have to acknowledge the nationally disgraceful fact that there is not a public laboratory in the entire country devoted to this purpose in the interests of public health. I mean laboratories supported and controlled by the governments, state or national. On the other hand, two states had inaugurated this work as early as 1886, Nebraska being the first to get into the field, while Missouri was the second, and attempts have been made in the same direction by the Agricultural Department at Washington, all looking to the recognition of one side of the other half of a cardinal principle, viz., the protection of a portion of the public wealth. This idea has received still further endorsement by the so-called "Hatch Bill" fund. But this is all to protect animal life from disease. Not a dollar for man! With regard to him, its millions of lives as a tribute to the grim monster disease, but not a dollar for defense.

The trouble is, man represents no value whatever in the eyes of a government that has little or no need of him for defense. In the interests of the development of measures for the protection of the public health, the very necessities which make it imperative to value its people as so much possible food for the hungry cannon, make it also imperative upon governments so situated as to require standing armies to provide a surplus of that food, and to keep it in the best possible condition ; for upon that depends the existence of such governments and the nations they represent.

The question of the relation of a standing army to a nation is not all negative by any means. The conditions which render them necessary are a blessing to any country. The country where they are internal is more fortunate than one where they exist externally. The continued existence of danger to the great national interests or national integrity, is an absolute necessity to the advancing development of a nation and the continuance of a creditable and intelligent system of government.

So where man is valued by the government, it is because he represents a factor in the national preservation. But not only does man represent such a factor, but everything related to his welfare, the crops he raises, the animals he uses in labor or for food, the natural resources of the country out of which he makes the necessities and even luxuries of life, are all necessary factors which demand the watchful eye of the government that the resources of the nation may be continually kept at their highest flood.

Recognizing, then, all these necessities, live governments have looked about and asked by what means can all these things be attained to the fullest degree possible?

There can be but one answer !

By continued scientific investigation and the application of the results to the needs of the people.

In other words, the

PRESERVATION OF THE PUBLIC HEALTH AND THE PROTECTION
AND AUGMENTATION OF PUBLIC WEALTH

Fulfills the obligations of any government to its people. This is the only standard by which to judge a government. Try the American by that standard. Form amounts to nothing ! The result, according to the standard, is the test, let it be a monarchy or republic.

Monarchies have been the first to recognize, as well as the most successful executors of this fundamental principle. It was not that they happened to be monarchial governments, however, that led them to do so. Location—the fact that the existence and prosperity of the government happened to be the same thing as the existence and prosperity of the nation has been the cause. France recognizes this principle to-day as much as Germany, and no European country can afford to neglect it. We think we can afford to! We have thus far neglected the principle, but it is encouraging even that the most mercenary of motives have led to the recognition of a part of it. Some day human life may have value enough in the eyes of our government to stimulate it to support scientific research for the protection of the public health as a humane principle, if not as public necessity.

Now what can research do in this regard? It can find the nature and causes of the diseases of life, and arrive at the means to prevent, or better, control them. It can discover new materials in the earth and new uses for old materials, or derive new ones from materials now thrown away. Its value to a country can never be measured by money, nor can its value be calculated by the results of any one man's work. It is the cumulative work of all workers which shows the true value of scientific research to a country or to the world. It is a fact that the results of scientific research are generally cosmopolitan in their far-reaching effects. The scientist should never feel that he belongs to any state, country, or people. "The world is my country and to do good my religion," should be the soul-inspiring principle of the scientist. A country, a people, should be proud to claim him as theirs, but the world only can reward his memory. Limit Jenner to Britain if you can! Confine a Virchow, a Darwin, a Helmholtz, a Humboldt, a Franklin, a Koch, or a Pasteur within the narrow limits of a country. Like Monte Cristo, "the world" is theirs. The same is true of every great defender of human rights.

Hence, scientific research must be supported and controlled by the respective governments in order that the best possible results may be obtained.

Fitness for work is the one essential in the worker! Politics, social ideas, or any other ism, should not weigh an iota in selecting men to fill positions the results of which may bless humanity from pole to pole, from the rising to the setting sun.

To the best interests of the public health and protection of our animal wealth from disease, original research must be supported and controlled by the government, state or national. This is not the case at present. We have no really scientific center of learning and investigation which we look upon with national pride as a government institution. We have a few laboratories in the East established by the endowment of human vanity through the urgent begging of the faculties of private, speculative medical schools, whereby the weaknesses of several wealthy men were taken advantage of in order to increase the annual incomes of their faculties. These laboratories are resonant honey-pots set to catch flies (students). The Carnegie laboratory occupies a big building with elegant fittings and much assistance. Two other medical schools in New York have similar advantages. One was endowed by the wealth of the Vanderbilts. With all their money, and though in existence some years, not all of them together have done any creditable work. Oh, I forgot, it is reported that some very scientific work has emanated from the Vanderbilt laboratory. Some poor, little, frozen up bacteria were found in ice. How thankful they must have been when thawed out. From time to eternity endowed institutions will never be any blessing to a country as nurseries of science. They are nepotistic hot-houses! No one but their own graduates, and, as far as possible, such as are related to a favored few, or mere tools willing to bow down to the idols of an imaginary "cultchaw," ever have any opportunity for employment in these endowed institutions.

Native ability, born out of the blue, has no chance!

On the other hand, the curse of America enters our public institutions.

Our public institutions are unfortunately very liable to suffer in the quality of those who are deputed to control and watch over their welfare, in comparison with the older endowed institutions with a large and alma-mater-attached alumni. Whatever may be objected to such institutions from their nepotistic-alma-maternal tendencies in the selection of instructors and workers, the fact that their overseers are generally elected by the alumni, guarantees the selection of men of high calibre. The evil is, that their entire energies are bent towards the welfare of the institution itself, instead of the institution as an integral part of the state and its relation to the same.

Our state educational institutions suffer in this respect in comparison. The overseers may be either nominated by the governor, or be elected by the people, it matters not which, the cardinal point of selection is, that those nominated, or elected, must be adherents of the prevailing political creed, instead of, are they the broadest minded, the best educated, the most patriotic men, as regards the welfare of the state, that can be found? It seems to me that the most honorable position in any state must be that which is responsible to the state for the higher education of its youth. This responsibility does not limit itself to-day, or to-morrow; the work of such men will not be judged by the present alone, "but after many days." If the policy of our endowed educational institutions is narrow, how much greater is the danger of our state ones being still more circumscribed. To a measure, the political lines are not drawn very closely as regards instructors; but that they can be, may be seen by the recent prohibition experiences in Iowa. To the best interests of the state, neither politics nor any other ism should have a particle of influence in the selection of instructors or the guidance of instruction itself. Fitness for the work demanded is all the state requires. Absolute faithfulness to that work is the only standard of judgment. The worker is equally responsible to the state, in that direction, with those who selected him. Insubordination is impossible, except insubordination to the faithful carrying out of these obligations to the state. The instructor, or worker, should be free as the air, so long as he fulfills his obligations to the state. The petty controls and tyrannies which the directing power of endowed institutions frequently exercise are entirely out of place, and must be rendered impossible with reference to the workers and instructors in state institutions. Nepotism may permit that in an endowed institution which would be insulting to a worker who is a servant of the state.

While eminent respectability, coupled with mediocre ability, is the general characteristic of those filling scientific chairs in our endowed colleges, still, the worker has one great advantage over his colleagues in state institutions. His commonplace respectability guarantees his continuance in his position, regardless of his mediocre ability. In state institutions, on the other hand, the utmost devotion to public interest, and the most extreme scientific abilities, will not be found sufficient to continue a person in position against unprincipled poli-

ticians, jealous interference, or the personal spite of those sometimes in authority.

Security in position, so long as a person has the ability required, and honestly devotes himself to the public interests, is an absolute necessity to successful scientific work.

As is easily to be seen, neither our endowed or state institutions offer conditions favorable, nor encouraging, to those who desire to serve their country as workers in the field of original research.

Original scientific research has not yet found a permanent resting place in either Great Britain, the United States, or in any country where the English language is spoken.

The ambition of my life is and has been to remove this disgrace from the people of this country.

It can only be done when our governments recognize the principle that

THE SUPPORT AND CONTROL OF SCIENTIFIC RESEARCH IS THE ONLY FOUNDATION UPON WHICH THE AUGMENTATION AND PROTECTION OF THE NATIONAL WEALTH AND PRESERVATION OF THE PUBLIC HEALTH CAN BE BUILT.

AN APPEAL FOR A NATIONAL LABORATORY.

As said, Science is the search after the facts in nature and the endeavor to correctly interpret the same.

Science is the only true democracy on the face of the earth. Genius is born. It cannot be made. It is as liable to find its birth-place among the poor as the rich. It finds its origin in the hovel, clad in rags and filth, as often as in the palace and arrayed in the robes of a Solomon. It is the only aristocracy. The possession of brains elevates the individual above the masses in every calling of life. The science with which we have to do is that of Pathology. Its mission is to discover the causes and nature of disease. It lays the foundation of all preventive medicine. In this sense Pathology is general, not special. When rightly pursued it knows neither man nor animal as a special creation. It recognizes life only; hence I have tried to make my work as applicable as possible to the diseases of human life, as well as valuable to the stock-raisers, for whom it is especially undertaken.

Genius thus being democrat in origin, and science equally so in its development and results, it is evident that the country must not—can-

not—wait for, or rely upon, the sons of the rich for its development in this direction. Like the rains, the blessings of scientific research, the results of preventive medicine, fall upon the rich and the poor alike. Disease is no respecter of persons. The health of our people is the very nucleus of our national wealth and progress.

With these facts in plain view it is self-evident that the one thing to do is to

ESTABLISH A NATIONAL LABORATORY AT WASHINGTON IN THE INTERESTS OF OUR PUBLIC HEALTH, AND THE NATIONAL WEALTH INVESTED IN LIVE STOCK.

Who will be that man of all men, that statesman of all statesmen, that Bayard of the nineteenth century, the knight beyond compare, to bless this country in especial, and the world in general, by placing a bill before Congress for this humane, benevolent, and economical purpose?

Mr. Hatch, will you increase the blessings the country now owes you by assuming this task?

Congressmen of Nebraska, will you show that you possess the large-heartedness, the energy, the practical common sense attributed to the sons of the West, where, though the sun may sink, still the sons of man are developing the country as never before, by inaugurating this work, which shall be an economical blessing to those interests upon which the country you represent largely depends, and for which a nation of humanity, suffering from preventable diseases, shall bless your names forever?

You are ambitious! You want glory! You would gladly have a nation build a monument to your memory! Here is your opportunity—the grandest ever offered to a citizen of this country!

Genius sees the opportunity and makes it its own.

Build your own monument!

One word as to where to place it in order that all purposes may best be fulfilled and the most intelligent control exerted.

IT MUST BE PLACED UNDER THE U. S. MARINE HOSPITAL SERVICE.

There and there only can we expect the nation's demands to be carried out.

The Agricultural Department has been tried and found lamentably

wanting. Let it stick to its regulative and preventive work. Disease is a unicum. Life is one. All scientific research in this direction must be so regulated as to bless and protect man's life as well as his pocket. Such an institution must be absolutely democratic. It must be a place where the poorest can get an education in these lines; where those without the means to work, but the fires of genius to do so, can find opportunity, with room and material free.

In this way only will our government ever be able to show that it realizes its first duty to its people. In this way only can we inaugurate and make practical that cardinal principle of national existence,

THE PROTECTION OF THE PUBLIC HEALTH; THE PRESERVATION
AND AUGMENTATION OF THE NATIONAL WEALTH.

This done, the United States will be able to take her proper place in the congress of intelligent and progressive nations.

The first really important move towards the establishment of scientific research in the United States was that made by the Hon. William H. Hatch in 1887, when, through his agency, the so-called "Experiment Station Bill" became a law of the land. The sum appropriated, \$15,000 annually to each state fulfilling the requirements of the law, is as ridiculously inadequate to the work which comes under the compass of the law as seems to be the cerebral ability of the state authorities to comprehend how the work should be done. But if those entrusted with conducting this work will only take advantage of the liberty of selection which the bill allows, and concentrate their energies upon some one object for a term of years, something valuable may eventually result. The example partially set by Nebraska can well be followed in this direction, though even here the influences of selfish ambition are endeavoring to spread out the work in several ridiculously useless directions, and unless vigorously combated, will succeed in hindering the development of that branch of investigation which the state especially requires.

That portion of this bill which especially interests us reads as follows:

"SECTION 1. Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled: That in order to aid in acquiring and diffusing among the people of the

United States useful and practical information on subjects connected with agriculture, and to promote scientific investigation and experiment respecting the principles and applications of agricultural science, there shall be established, under direction of the college or colleges, or agricultural department of colleges, in each state or territory established, or which may hereafter be established, in accordance with the provisions of an act approved July 2, 1862, entitled "An act donating public lands to the several states and territories which may provide colleges for the benefit of agriculture and the mechanic arts," or any of the supplements to said act, a department to be known and designated as an "agricultural experiment station." Provided, That in any state or territory in which two such colleges have been or may be so established, the appropriation hereinafter made to such state or territory shall be equally divided between such colleges, unless the Legislature of such state or territory shall otherwise direct.

"SEC. 2. That it shall be the object and duty of said experiment stations to conduct original researches or verify experiments on the physiology of plants and animals; the disease to which they are severally subject, with the remedies for the same; the chemical composition of useful plants at their different stages of growth; the comparative advantages of rotative cropping as pursued under a varying series of crops; the capacity of new plants or trees for acclimation; the analysis of soils and water; the chemical composition of manures, natural or artificial, with experiments designed to test their comparative effects on crops of different kinds; the adaptation and value of grasses and forage plants; the composition and digestibility of the different kinds of food for domestic animals; the scientific and economic questions involved in the production of butter and cheese; and such other researches or experiments bearing directly on the agricultural industry of the United States as may in each case be deemed advisable, having due regard to the varying conditions and needs of the respective states or territories.

"SEC. 3. That in order to secure, as far as practicable, uniformity of methods and results in the work of said stations, it shall be the duty of the United States commissioner of agriculture to furnish forms, as far as practicable, for the tabulation of results of investigation or experiments; to indicate, from time to time, such lines of enquiry as to him shall seem most important; and, in general, to furnish such advice and assistance as will best promote the purposes of this act. It shall be the duty of each of said stations, annually, on or before the first day of February, to make to the governor of the state or territory in which it is located, a full and detailed report of its operations, including a statement of receipts and expenditures, a copy of which report shall be sent to each of said stations, to the said commissioner of agriculture, and to the secretary of the treasury of the United States.

"SEC. 4. That bulletins or reports of progress shall be published at said stations at least once in three months, one copy of which shall be sent to each newspaper in the states or territories in which they are respectively located, and to such individuals actually engaged in farming as may request the same, and as far as the means of the station will permit. Such bulletins or reports, and the annual reports of said stations, shall be transmitted in the mails of the United States free of charge for postage, under such regulations as the postmaster-general may from time to time prescribe."

We see that it provides for "conducting original research" into the nature and causes of diseases of our domestic animals, and also permits the state authorities, or those entrusted with the general supervision of the work, to concentrate their means upon any special interest in their respective states. The conditions in our western states are peculiar in comparison with the majority of those situated east of the Mississippi river. Nebraska is no exception to the rule! Her financial prosperity and future development depend upon her live-stock interests. Though her people have more liberally provided for the protection of those interests than those of any other state, in no state have the results been less commensurate with the means provided. Nothing but the utmost vigilance on the part of the live-stock men will prevent the means given by the Hatch bill being wasted in the bloom of useless agricultural experiments, the value of which is altogether too well known to the people from past experiences. This live-stock basis is of a perishable nature, and very liable, in fact constantly so, to excessive variations from the ravages of pestilential diseases, the chief of which has been, and is, the swine plague. In fact, the future development of these states, and the prosperity of the immigrant farmer, are largely dependent upon the success of the hog crop. There are other diseases, however, which will yet make themselves as unpleasantly felt as the swine plague. Among them are two cattle diseases which we are now engaged in investigating. The work which this and my former report represents was instigated through the live-stock men of Nebraska, represented by the State Board of Agriculture, coming to a realization that these great interests were constantly being imperiled through the ravages of devastating diseases. Danger here, as everywhere else, was the stimulus which led to the inauguration of this especial work. Whether the work thus far done has been such as to warrant the approval of those directly

interested, they and the reader can best judge. Every promise which these researches intimate will eventually be fulfilled, if the work finds sufficient support to carry it out. As has been said, this work was begun July 1, 1886, and in the very first case of swine plague which came to my notice, after I was enabled to even begin investigations, the germ was discovered, and no other organism than the one then discovered has ever been seen in sick hogs during our researches. The ease with which this discovery was made makes it entirely incomprehensible why so many mistakes and so many different organisms have been, said to be, discovered by the authorities at Washington. We had to begin in the very smallest manner, and except the laboratory fittings, which were my personal property, until April, 1887, we had neither means nor suitable conveniences to carry on work of this kind. As other states may think of engaging in this branch of research, it may be well for me to detail what should be the appointments of a patho-biological station for the study of animal diseases, and to enumerate some of the difficulties under which I have labored.

The greatest difficulty one has to contend with is, that our governing bodies are so utterly inexperienced and often so uninterested in this kind of work, that they fail or do not try to grasp its many intricacies, and especially the detailed expenses which its requirements necessitate, or the time it takes before practical results can be attained. They cannot, or will not, see that Science is the search after facts and their correct interpretation. The application of the results belongs to another field than that of original research.

Scientific research is expensive. When undertaken, as has been the case here, without the necessary means or suitable conveniences, any success can only be attained at the imminent risk of the health of the worker, for nothing but the most determined and obstinate persistency, and a thorough devotion to the interests of the state, could ever have led to the results which have thus far been realized. For the edification of my European confrères I will say that, during the time which is represented by the work herein published and my former report, I have been "maid of all work," washing every glass, keeping my laboratory clean, preparing everything used, making every experiment, traveling over the state and never having an iota of assistance that amounted to anything. This very report was written, from my

notes, in four weeks, and beside the germs described in this and the two in my former report, two other unrecognized diseases would have been worked out with equal exactness, and we should be now ready to begin reports on them, had we had any suitable means to work with; and beyond that, the question of swine plague inoculation (though belonging in the field of hygienic and preventive medicine and not mine), which should have really been entirely completed so far as a patho-bacteriologist can do it, would have been done if my work had not been constantly obstructed by unnecessary interference.

We had to begin, unfortunately, at the wrong end of the rope. We began to work without a proper place to work in. The first thing essential is a suitable piece of land; say, not less than five acres. Ten acres will be found ample for any emergency. The land is the easiest acquired of all these necessities; especially in our western states. The land, however, is not the all, by any means. Such a station being for the experimental study of diseases of a dangerous or threatening character, as regards the live-stock of the state, it is an absolute necessity that it be looked upon and treated as a quarantine station. It should be at once fenced in with a tight board fence, having suitable gates that should be invariably kept locked.

When once the buildings are erected and the regular work commenced, no one should be allowed entrance except with the permission of the person entrusted with the work of experimentation. No one else should have the privilege of entering such grounds, or of giving permission to any outside person. Under all circumstances the greatest precaution must be exercised against the admission of strangers.

So far as this station is concerned, about all we had was the land, and even with the assistance of the "Hatch bill" fund that is about all we have now, except the laboratory fittings, which are more complete than those of any other laboratory of the kind in the country.

In order to do this work properly, it should not be begun as we had to do in Nebraska. The first thing should be the setting apart of the land, the fencing of the same, and the erection of the necessary buildings. There should be a building for the small animals necessary to experimentation, of which there should never be less than 500 on hand at all times, besides several hundred more

breeding stock to keep up the supply. This includes rabbits, guinea-pigs, white mice, rats, ground squirrels, kittens, and dogs.

Notwithstanding every endeavor on my part, it has therefore been an absolute impossibility to collect the necessary stock of this character to do the work demanded.

There must also be buildings, sheds, and yards for keeping the larger animals, also necessary for experimentation, but not in immediate use, as well as a regular experiment building for the animals experimented upon, which should be distant from the others, and really ought to be fenced in and form a quarantine within a quarantine. Aside from all this there must be the laboratory and work building fitted up to meet every requirement that can be in any way necessary, and equally essential is a residence for the investigator and one for the servants, for this work is of the all day and all night kind.

Now, scarcely one of these things have we had, having up to the past winter but one small room for work and six movable wooden hog-pens. For the want of these things my work has scarcely been advanced an iota since January, 1887.

What can be done with \$15,000 towards the establishment of such a station, especially when salaries and everything else, including agricultural experiments, must come out of that small sum? The allowance of \$3,000 the first year for building purposes, should be made \$5,000 per year for a period of ten years, to be used as the authorities see fit in accordance with the law.

As it is to-day, the University of Nebraska and myself have about \$20,000 already invested in this one branch of work, and all we have is an insufficient building and no laboratory building, and but the fittings of a laboratory and my library. Without a library of reference, continually kept replete with all new publications, this work cannot be prosecuted. This requires an outlay of not less than \$1,000 a year. We have already three rooms occupied and an autopsy building suitable for small animals, if very little experimentation is done. These rooms consist of a work room, a cultivating room, and a study. Out of the "Hatch-bill" fund we shall get a small building for the larger animals necessary in experimentation, capable of easy cleansing and disinfection, with Portland cement floors, and covering to the brick walls and partitions. It will be fitted with a small number of pens for swine, and larger ones for cattle and horses, and

cages for dogs; for, out here we have considerable of a something called "Rabies," but as yet have been unable to touch it because it could not be done with safety. This building will be supplied with a crematory large enough to burn up a hog, and a small work room. But when I say it is two miles from our work rooms, and that three such buildings are an absolute necessity, as well as a laboratory building, all of which should be together, the reader can at once see that experimentation can only be done at great inconvenience and much waste of time, and that during the severe weather of winter and the times when our roads are almost impassable, that these obstructions to good and continued work will be frequently almost insurmountable.

It is self-evident that unless each state takes active hold of this matter, that very little of practical value to the live-stock interests can ever come out of the small sum of \$15,000 appropriated annually by congress, if that body does not see fit to increase it. Then, again, there is plenty of evidence that the bodies governing this work in many states have at once lost sight of the cardinal principle of good work. Concentration of means and energy! So far as the bulletins yet issued indicate, Nebraska is the only station which is giving any especial attention to live-stock matters. Those of Texas, Kansas, Missouri, Arkansas, Colorado, and other stock raising states should do the same. The managers of this fund should realize that the whole thing is an experiment. Hence, would they insure its final success they must concentrate their energies upon such questions as will bring the best practical results in the shortest space of time. The American public is the most unreasonable despot on the face of the earth. It wants results even before work is fairly begun. While my natural bias may make me one-sided, still it is self-evident that so far as the great live-stock states are concerned, there is but one field of operations open which promises any permanent and reasonably immediate practical results, and that is the study of our animal diseases and the feeding and conditions favorable or unfavorable to live-stock, not only with reference to preserving it from disease, but also for the better protection of the public health. To waste valuable means in useless agricultural experiments under these circumstances is to criminally neglect the great interest of such states.

These remarks, while but very insufficiently showing the difficulties

which surround and oppose original research, will also, it is hoped, prove both suggestive and instructive to the authorities of other states.

THE HYGIENIC PREVENTION OF SWINE PLAGUE.

No matter what may be the final results of the inoculative prophylaxis in relation to many diseases; no matter if it eventually equals in trustworthiness and safety vaccination in small-pox—still, as in that disease every restrictive measure has to be vigorously enforced in order to check its ravages, so, in order to keep the losses from swine plague, and other diseases which may be so treated, down to the lowest possible level, the state and the animal owner must unite in the most exact execution of all those preventive measures which daily experiences and scientific investigation have shown to be practical and valuable. It may somewhat surprise the lay reader to be told that some of the chief causes of swine plague must be sought in the inattention, carelessness, or want of intelligent action on the part of owners themselves.

To speak plainly, and that is in accordance with my obligations to the live-stock interests of the country, these human causes are:

- | | |
|-----------------|--------------------------------------|
| 1. Ignorance | } on the part of the owners of hogs. |
| 2. Laziness | |
| 3. Carelessness | |

Let us consider them in their order.

1st, Ignorance. It is sometimes well that we "scientific fellows," "those theoretical chaps," should have the floor, for it has generally been the case that the other side have had the most to say, and one of their chief remarks has been something like this: "Yes, yes; your laboratory work may be all right; I think it is; but the trouble is with you fellows you do not have any practical experience; when you have seen as much hog cholera as we have, then you may be able to tell us farmers something about it."

Now, any one who has read the foregoing pages of this report can see that these suffering farmers really have some just grounds to talk as they do. Have they seen that "the demonstration of the contagiousness of this disease has enabled our agriculturalists to do something to prevent its spread"? (Salmon, 1883, l. c., p. 56.) Do they not know that in 1888 they are not a bit better off in this regard than

they have been? Swine plague is still as rampant as ever, notwithstanding Mr. Salmon's promises!

Hence, I think these "agriculturalists" have some right in their flings at us "laboratory fellows," although it is somewhat unjust that all should suffer for the sins of omission and commission of one single individual. Had Dr. Detmers not been more successfully "stamped out" than the swine plague, I have no doubt that "our agriculturalists" would have long since derived some benefits from the results of scientific research.

But let us see if the tables cannot be turned upon these "practical fellows" in spite of their many years of "practical" experience. Now, gentlemen, you have been over forty years gathering in experience! How much do you know about swine plague? Have you learned the very first lesson which such vast experience should have taught you long ago—were you the "practical" men you fondly believe yourselves to be?

Every one of you knows that a contagious disease practically means that the sick animals are directly dangerous to healthy ones. The agricultural department of Washington has been telling you this for the past ten years, though it contradicts its teachings when it tells you that the places where sick hogs are or have been may be dangerous, because the germs can develop in the earth and continue to live there. What plants the germs there? The sick hogs, with their manure and urine!

To be sure, they are still in the dark how long they can live there. They tell you "it can multiply and live (there) for a limited period," but I have given you testimony, such as no scientific experiment can excel, practical testimony such as many of you can corroborate, that it can live in the earth, in pens, in refuse, where sick hogs have been, for one year, two years—aye, three years—and never a hog, sick or well, be in the place in the meantime, and still the swine plague has broken out in healthy stock when put in such places. The case at our "college farm" cannot be contradicted! The record tells us that the last hog to die there in the former outbreak was on May 4, 1885, and the next time the disease broke out, in the same hog-house, was December, 1887. During that time no swine plague diseased hogs went near that place, and in my experimental hogs there had not been a case since August, 1887, and from that time until

to-day—June 30, 1888—not even a pig among my experimental stock has been sick. Now, as no sick hogs were in that pen for over two years and a half, and as my hogs were and are well, where is the chance for contagion to have got in its work? It is impossible! Still, with such experiences staring you in the face, you have gone on getting more and more “practical” (?) You have had your hogs all die; you have killed all the sick, and then sold the balance; you have let your pens lie empty for months, if not years, at a time; you have lost your courage, and have regained it; you have bought unquestionably healthy stock and put it into those old, uncleaned, and litter-covered pens—and with what result?

Sooner or later swine plague has broken out!

That it could not possibly be due to contagion should have been evident to you from the fact that had it been it would have broken out in 8 or 10 days after you purchased your hogs; on the contrary, weeks and months have gone by, and even though you have not bought another hog, even though all your neighbors have been free from it, still swine plague broke out in your hogs.

Then you went over the same old practical experience. You sold off or killed off all. You waited awhile; bought more, and again you went through the same experience.

Did you learn anything thereby? Did you get that “practical knowledge” you boast so much about? Did you learn that those pens, that litter, was the source of danger?

No, you did not! Then why talk to us “scientific fellows” about “your practical experience”? Were we so incapable of appreciating our practical experiences we should indeed be willing to acknowledge the corn, and you could tell us, as I do you, that willful “ignorance” on your part has been one of the chief reasons that you suffered unnecessarily from the swine plague.

You have not done as well as we have!

Again I say, you have folded your hands; shut your eyes; refused to use your brains; absolutely ignored the most positive lessons of experience and gone on cursing and lamenting your luck; given it up for a time; purchased new hogs in the end, and again gone through with the same bitter experiences, and again and again refused to see the plainest fact that it would seem a blind man should gain a knowledge of; you have still failed to see that the places where sick hogs

had been, or were still, did and do remain centers of danger for a long time.

I have told you that Mr. Salmon sometimes advises better than he theorizes, and that, like most other observers, he contradicts his assertions as to the nature of a disease, when he comes to write about preventing it. Now, though he tells you that swine plague is a "contagious fever," he advises you entirely contrary to that assertion, for no contagious disease germ ever known deports itself in the way Mr. Salmon's directions indicate. Nothing but the germ of an extra-organismal infection can keep its vitality in such materials and under the circumstances mentioned in the two following quotations.

"1. The regulations should go into effect in winter or early spring, when fewest animals are affected, or when, as my experience indicates, the disease is entirely extinct."

"2. People living in localities where the disease has prevailed should keep their hogs in an enclosure free from accumulations of manure, straw, litter of any kind, or remains of dead hogs, in which the contagion might possibly be preserved, and in which there were no sick animals the preceding year." 1883, l. c., p. 442.

Swine plague is a local infectious, and not a contagious disease.

Now, having taught you the real nature of swine plague, it will be your fault if you are not more successful in preventing it in the future.

The question is, will you farmers do it?

This brings me to my second accusation:

Laziness.

It may appear wrong in me to lay this fault at the door of you farmers, but the truth compels me to do it. Many of you are really too lazy to do the necessary work to prevent hog cholera. The writer speaks from actual personal experiences. Where a very few well informed farmers felt it "too much trouble" to separate their well from their sick hogs, and use ordinary precautions in the feeding and care of them. The separation was done, however, and with the most surprising results. Not one of the well hogs selected and separated from the sick lot and placed on fresh land became sick. This treatment has been carried on under my direction in numerous other instances with the most satisfactory results.

Is it too much to ask you farmers to make a test in every case,

where you are the sufferers, of this separation and isolation method. It requires some care, a great exactness and observance of a very few little points, upon which depend their success, however.

Lazy farmers will never prevent hog cholera. Men too indolent to observe the fact that their pens are the chief source of danger, when the fact has been before their eyes for years, will never prevent it—unless the state makes them. Men who are too lazy to prevent by isolation and care would have hog cholera if we had an absolutely perfect vaccine. Such men would be too lazy to have their hogs inoculated even if the state did it for nothing. They would wait until their hogs became sick and then cry out for inoculation, which, if it were done, would be too late to have any effect. Then these very men would be the loudest to condemn the method and the extravagance of the state in keeping up such a costly institution.

The next point to call attention to is,

Carelessness.

In order to prevent hog cholera, constant care and watchfulness is necessary not only with regard to one's own hogs, but as to what is going on among his neighbor's hogs. It will not do to isolate the well from the sick alone, but all contact between the places where these hogs are must be entirely avoided. The place where the sick hogs are, or were, must be treated as a pest house. Well hogs must be kept not only from it, but also from any possibility of contact with any straw, dirt, manure, or other material in or from any such pestiferous place.

Hog cholera is an infectious disease. The chief and only real seat of danger is where sick hogs are or have been.

The sick hog is dangerous only because he can infect other places. These places thus become pestiferous to healthy hogs.

RÉSUMÉ.

1. *Don't* leave a well hog in a place where a sick one is or has been a moment longer than can be helped.
2. *Don't* fail to examine such separated well hogs twice a day, and to remove any that may become ill from the others.
3. *Don't* allow the same person to take care of the affected and well hogs.
4. *Don't* allow any intercourse of men, dogs, or hens between the pens of either lot of hogs.

5. *Don't* put a new lot of healthy hogs in a pen, or upon land, where swine plague has been for less than three years, unless the same has been thoroughly cleansed of all refuse, ploughed, or dug up several times, and exposed to the air for an entire summer season.

6. *Don't* forget that closed pens, sheds, straw stacks, and accumulated litter are more dangerous than open country, when swine plague has prevailed in such places.

7. *Don't* water hogs from running streams.

8. *Don't* place your hog-pens, or runs, so that they can drain into running streams.

9. *Don't* forget that all such places should be well drained and kept as dry as possible.

10. *Don't* bury dead hogs when you can burn them up.

11. *Don't* sell or buy sick hogs.

12. *Don't* visit your neighbor's hogs when sick or allow him to visit yours if well.

13. *Don't* forget that watchfulness, carefulness, and diligence will do more to prevent swine plague than all medicines.

14. *Don't* forget that without these things being adhered to the most practical vaccine will ever prove next to useless.

15. *Don't* forget to keep to these rules!

REGULATIVE PREVENTION OF SWINE PLAGUE BY THE STATE.*

WHEN SWINE PLAGUE EXISTS IN ADJOINING OR DISTANT STATES.

In this regard there are two points to be met:

First, swine plague introduced by infected swine from other localities.

Second, swine plague introduced by means of uncleansed and non-disinfected railroad cars or other conveyances the property of common carriers.

Let us consider the first condition:

All importation of swine, either for stock or feeding purposes, whether or not such imported swine came from states or localities in which swine plague exists, or has existed, should be prohibited by law, unless the state provides suitable and safe conveniences for the absolute isolation and quarantining of such imported hogs, at selected

* Penalties must be attached to such regulations, but as this is a matter for legislation I have not considered it my duty to consider it.

points of entry, so that each lot can be safely and securely confined in a previously cleansed and disinfected hog-pen.

In all such cases the state would be liable should the hogs become infected while in the quarantine pens, if they had not been previously cleansed and disinfected, or on account of neglect of duty on the part of any servant of the state. In order that the quarantine hog-pens constitute a safe and suitable place for such imported hogs, it is absolutely necessary that they be individual pens of various sizes; that the walls be of the hardest bricks, and the gates of iron, and if any portion of them be roofed, that the same be of iron also, and the bottoms made of cement, not pitch, asphalt; the feeding troughs should be of stone or iron, and of such a size as to be convenient to handle and cleanse. The pens should be at least ten feet apart, the passage ways between them being of cement asphalt and so graded that the drippings or washing from each pen could not run over to the opposite pens. The entire quarantine grounds should be sewered. The drainage from each pen should enter the sewer from a trap within the walls of the pen so that in washing them no material could get outside of the pen.

The quarantined hogs should be inspected daily, by a competent veterinarian, for a period of twenty days subsequent to their arrival upon the state territory.

If no evidence of swine plague or other suspicious disease occurred during that period, the hogs should be declared free and released to the owner.

The state should take such measures as would insure their further transport into the state in cars or conveyances that had been previously cleansed and disinfected, whether said cars had been used for the transport of swine or not previously. In such cases every means of transport must be looked upon as "suspicious" and treated as such.

Should the swine plague break out in a lot of imported hogs while thus quarantined, the pen in which they were confined should be washed and disinfected daily, and every bit of straw and refuse carefully removed in iron hand carts, made for the purpose, and then burned in an oven provided for the purpose, and for the cremation of dead hogs. Great care should be taken that nothing be dropped from such a cart.

When the outbreak had come to an end, the quarantine should be

extended for a period of at least thirty days from the time the last sick pig had died, or entirely recovered from the disease. In such cases the animals should be thoroughly washed before leaving the pen. They should be conveyed from the quarantine in cleansed and disinfected cars as aforementioned.

Upon arrival at the owner's premises, he should be compelled to give them a temporary quarantine of still another twenty days before being permitted to place them among other hogs upon his place.

Whenever a lot of swine plague diseased hogs were thus quarantined, the chief inspector should detail one person to take the exclusive care of such diseased hogs, and absolutely forbid such a person going near, or having anything to do with other healthy lots of hogs that might be in the quarantine at the same time, or with any hay, straw, feed, or utensils used for the healthy hogs.

The persons having charge of the swine plague diseased hogs should be provided with separate buckets and utensils necessary to their care, and with hay, straw, and feed kept in a special place as near to the diseased hogs as possible.

He should also be provided with a suitable disinfecting solution. Corrosive sublimate in water, one to 1,000 parts, to wash his hands and boots each time he had been busy about such diseased hogs; said disinfectant should be kept near the pen in which the diseased hogs were confined.

The state should fix the price per head for hogs thus quarantined, and should forbid the inspectors or servants from charging owners any extra fees.

SWINE PLAGUE INTRODUCED IN TRANSIT.

Could we be absolutely certain that swine purchased in other states were procured from places where no cases of swine plague had occurred during the previous twelve months, still we cannot be assured that the disease may not break out in such swine shortly after their arrival in the state on account of their being transported in cars in which swine plague diseased hogs had been previously conveyed.

[A ranchman in the western part of Nebraska purchased a large lot of hogs from places where no swine plague existed, and where the disease has not since broken out among the hogs still remaining on the same places, but the disease broke out in the purchased hogs within

two weeks after their arrival at his ranch. There had never been hogs kept upon the land previously.

He was careful in selecting his cars, so he thought, and was assured by the agent that they had been previously used for conveying lumber. They were not swept out or washed or disinfected. He has since learned that some diseased hogs were conveyed in one of the cars to either Omaha or Lincoln some time previously.]

This fact, that buyers or shippers cannot assure themselves that no sick swine have been conveyed in the cars, not only renders a quarantine absolutely necessary for imported hogs, but suggests the necessity of the state organizing some method by which the cars used to convey stock of all kinds within the state should be properly cleaned and disinfected every time they are used for such purposes.

REGULATIONS FOR THE SUPPRESSION OF OUTBREAKS IN THE STATE.

All places where swine plague exists should be indicated by sign boards. Hog-pens or runs should, if possible, be upon high, dry land with a southern outlook, and so situated that they can be well drained and kept dry. Thoroughly wet runs, mud holes, are not as dangerous as such as half dry out in the summer months. Filth alone does not cause the swine plague, though it may support it. Too much wet and an excessive degree of dryness are both unfavorable to the continuance of life of the germ of swine plague in the earth, but our heavy, damp soil is exceedingly unsuitable to the development of a sufficient degree of dryness to be unconformable to the life of these germs. In a dry, sandy soil they should die out in the heat of the summer. Such hog-runs should never be so situated that the drainage from them could gain access to the wells from which the swine were watered or to running streams.

Owners of hogs diseased with swine plague should be cautioned and held responsible for any extension of the disease from their hogs to healthy hogs of other persons, when they failed to comply with the regulations to prevent the extension of the disease, such as:

1. The sale or removal of sick hogs or healthy ones from lots in which the disease existed. The sale or removal of any other domestic animals that had the run of such hog yards when swine plague existed among the hogs. At such times owners should be forbidden

allowing their horses or cattle (which were and are for work, or being daily driven over the public ways), to have the use of, or from being led into, or across the territory of such infected grounds. They should also be forbidden leaving their wagons or farm machinery in such infected hog-pens or runs, even though swine plague did not exist among their hogs at the time.

2. The removal of hay, straw, manure, offal, or earth from swine plague infected hog-runs or pens, or from such in which the disease had existed within the previous twelve months, and the conveyance of the same over any public way, or unloading such dangerous material anywhere along the same, should be forbidden by law.

(Owners of swine should use every precaution about distributing such materials over their own premises in any places where healthy hogs would be liable to come in contact with them.)

3. All hogs should be watered from wells.

(Watering infected herds of swine from running streams, or allowing them to wallow in the water of the same, should be strictly forbidden, as numerous cases are on record where the disease has been extended in this way.)

(The placing of rendering establishments on the banks of such streams, or where they could by any means drain into them, should be forbidden by law. This regulation should also apply to pork packing houses.)

4. Owners of swine plague infected herds or sick hogs should be forbidden to visit the hogs of other persons or allow such persons to visit their hogs, or enter the infected hog-runs or pens.

5. The farm dogs should be kept chained on farms where swine plague existed, and strange dogs shot if seen around such infected hog-runs or pens.

6. Owners should be instructed, or perhaps forbidden, not to put new and healthy hogs into hog-runs or pens in which swine plague had existed until all the hay or straw stacks that may have been upon the infected premises at such a time have been consumed by other animals, and the refuse remaining been completely burned up. Any pens, sheds, or stables in which or under which diseased hogs have been, must be thoroughly cleansed and the contents of the pens or sheds burned. Stables should really be so situated that the hogs could not run under them, or the drainage of the hog-pens settle un-

der them. When such is the case, the stable should be raised, the refuse under it be removed and burned, the bottom of stable whitewashed with lime and corrosive sublimate (one to 500 parts water); the earth forming the floor of hog-pens or sheds, or that under such stables, should be dug out for at least one foot and spread in a thin layer over the hog-run, the hole being filled with fresh earth from another part of the farm. The bottom boards of such hog-pens or sheds should be removed, loosened, the balance of the building to be whitewashed, as above, three times, and left exposed to the air for three months.

The ground of the hog-run or yard should be plowed up very deep and then harrowed three times, about a month apart, before any healthy hogs should be placed thereon.

(It will be easily seen that in most cases, here in the West, it will be safer as well as cheaper to place fresh hogs upon entirely new land with new fences and sheds, but where this cannot be done the above regulations must be exactly carried out.)

In case the hogs are placed upon new land, great care must be taken that no persons coming into relation with them, or any teams used to convey feed to them, cross the old grounds or come in contact with anything used among the previously diseased hogs. Tools, troughs, and such things used among the old hogs should be burned, and new ones provided for the fresh swine.

DISPOSAL OF SICK AND DEAD HOGS.

1. No sick or dead hogs should be removed from the infected premises.

2. Cremating or burning the carcasses of dead hogs until reduced to powder is by far the most reliable method to treat them.

3. Dead hogs should never be thrown into running streams, or so buried that the drainage from burial places could possibly gain access to such streams, or even wells or ponds used to water the stock.

4. Where cremation is impossible, and burial has to be resorted to, and the land is so situated that it can be done without conflicting with the previous conditions, a portion of the hog-run should be fenced off with a board fence, and a trench dug at least twelve feet deep and of corresponding length to a space that would be occupied by two-thirds the number of hogs present. The dead hogs should

then be at once removed and some crude petroleum poured over the carcass and the same covered with a layer of quicklime and six inches of earth. This should be done with each carcass. When the disease is at an end the entire mass should be covered with six inches of quicklime and the trench then filled up. No more hogs should be put into a trench than would allow of six feet of earth over the buried carcasses. The burial place should be kept fenced in for at least two years and sowed down with some strongly growing weeds or grasses. Where the situation of the hog-run is such that a suitable burial place cannot be made in it, or adjacent to it, on account of running streams or wells, such a place should be selected as near the hog-run as possible. The dead hogs should be carefully put in a wagon and never dragged over the ground when conveyed to any burial place. The wagon should be washed with a disinfectant each time after using, or else the owner might be wondering how he got the swine plague again if he purchased healthy hogs and complied with all the previously suggested conditions, if he conveyed them home in an uncleaned wagon.

I now come to the most difficult question of all to consider in connection with swine plague, and that is, under what conditions should the state be held responsible for its existence, and when should the state remunerate owners on account of the same, or any action of the state veterinary police authorities.

STATE REMUNERATION FOR HOGS KILLED BY THE AUTHORITIES.

The state should not be held responsible nor should owners receive any remuneration on account of any action of the state when an owner or owners failed to comply with all or any of the state regulations for the control of the disease, and swine plague broke out among his or their hogs.

Swine plague is an infectious disease already domesticated in Nebraska, hence the state should not be held responsible for what already exists where infected premises are one of the chief causes of the disease. It is not like Texas fever, which, although also an infectious disease, is still foreign to the state, and hence, if introduced, it could only be on account of the insufficiency of the state veterinary sanitary laws and executive force; therefore the state should be held liable for any damages resulting to domestic stock from it or any similar disease.

In this regard I want to say a word about pleuro-pneumonia and the responsibilities of the United States government in regard to the same.

While I fully believe that the government should be held responsible for its existence at all infected points at the present time, and should pay a large share, if not all, of the expense of stamping it out and to prevent its further extension, I believe it a bad policy to assume that the United States government would be wholly responsible for its further extension into the states west of the Mississippi, or that it should pay for all the damages accruing from such extension. The states in which that disease does not exist should be held even more responsible than the United States government for its entrance into their respective territories, and should pay their share for the damages accruing to ownership thereby.

If the whole responsibility for such extension and such damages be placed on the central government, the only result will be that the state governments will be lax in their obligations to their own people, and negligent in their duties in endeavoring to keep the disease off their territory.

With regard to swine plague, it seems to be but just that the state should be held liable for the full value of domestic hogs in all cases where the disease is introduced among them by imported hogs, or through the negligence of railroads or common carriers in not supplying properly cleansed and disinfected conveyances for such hogs.

Again, the state should be held responsible for all damages from swine plague when extended in the state through the negligence of such common carriers.

That such common carriers should also be held responsible to the state for such outbreaks, caused by their neglect, is beyond all question, but how much and to what extent I must leave to our legislators to decide.

Whether or not the state should be held responsible for the negligence of owners of diseased hogs in extending the disease by their not complying with such laws and regulations as may be made in the future, to prevent the extension of the disease, I will also not attempt to decide, but leave it to be discussed by legislators.

It must never be forgotten in considering these questions of remuneration and state responsibility, that the rights of the state must be as rigidly protected as those of the owners of affected animals.

Such are the means which practical observation and experience, guided by continued scientific research and a thorough understanding of the nature of the swine plague, suggest for its hygienic and restrictive control. The one question is, are they really practical? To which I answer most emphatically, "Yes!" Then comes the question, are they practicable? To which I also answer "Yes," but only under a government and with a people determined to execute them. In Germany they could be executed to the letter, and swine plague very closely controlled. The same result could be obtained in this country, but with governments that will not do their duty, and with people who do not seem to care to have it done, but had rather sit with folded hands and see their animals swept away by pestilence and wait for some all-healing panacea to be dropped, like manna from the clouds, the case is different. If the people are determined to help themselves, and will make stringent regulations and laws, and insist upon really competent veterinarians being appointed to see them executed, and place the actual executive agency in the hands of the local officers, sheriffs, county and town officers, and hold them responsible for the same, and make them subject to penalty, the losses from swine plague and other diseases of like nature need be no longer onerous to owners. Every hog owner must, however, do his duty as if he alone were the sufferer, and also conform to the "golden rule" so as to protect his neighbors.

THE PREVENTION OF INFECTIOUS SEPTICÆMIÆ BY INOCULATION.

In the following pages I do not intend to go into any detailed discussion of this question, from the fact that it is yet in its infancy, even in older countries, and again, I am not yet in a position to proceed with that exact experimentation which is necessary before one writes very much upon such an important subject. In justice to myself I must say that, except that the result of the second series of inoculative experiments made in 1887 fully confirm those of 1886, I have been utterly unable to proceed with this work for want of place and means, though I have used every possible endeavor to get them, feeling that the great porcine interests of the country demand it beyond almost anything else. I will also say, that continued and unnecessary obstruction has been put in my way, and every endeavor made to retard my honest endeavors and throw discredit on my work

by parties whose obligations to the state should have made them at least neutral, however unpleasant my existence in Nebraska may have been to them.

Let us consider the question of inoculation briefly from the historical point of views :

Vaccination and artificial inoculation, as the latter is understood, are two entirely different things. Both are dependent, however, on the result of practical observation. Both are dependent upon the fact that practical experiences have shown that, as a general thing, individuals that have survived an attack of a given disease are not open to a second attack within a variable term of years. This is not a law, it is simply a rule, and open to many exceptions, each of which have their special cause if it only could be discovered. The historical development of vaccination is much longer and older than the very modern idea of inoculation as a means of preventing infectious diseases. The practically gained knowledge, that one attack of a large number of strictly infectious diseases renders the individual immune against a second, is not new, but scarcely well enough established to be universally accepted. Vaccination, properly speaking, found its origin in variolation, or the conveyance of small-pox to healthy individuals from those already sick. So great had become the skill in this procedure, long before Jenner's time, that it was almost universally resorted to in order to cause a general extension of the small-pox in a community, and thus shorten the period of an outbreak as well as lessen the danger of its extension over the country; for this procedure was only resorted to under special quarantine regulations, the observance of which was carefully watched over by government officials. So great was the skill acquired in variolation by certain Italian and French physicians that they not only knew how to select those small-pox patients who would be most liable to transmit the disease in a mild form, but how to introduce the material so that this desired end would be the general result.

Then came the wonderful observation of the immortal Jenner, the greatest benefactor to the world that humanity has any record of. He observed that the dairymen and maids acquired local eruptions upon their hands, when wounded, who were busied about cows that had the disease known as cow-pox. He observed further that such persons escaped small-pox when it raged all around them. He in-

oculated from cows on persons, and then inoculated the same person with genuine small-pox, and proved the preventive value of the former against the latter. Hence it was that the cow-pox acquired the name *vaccina*, but a genuine cow-pox never existed. Cow-pox is nothing more or less than bovinated small-pox, which at some time in the unknown past was transmitted by small-pox diseased persons to cows, and thus from cow to cow until it acquired a certain constancy in character and a certain fixed mildness in virulence. The ordinary world is totally unaware of the fact that since the generalization of vaccination and its wonderful effects in the prevention of small-pox, that the disease known as cow-pox has almost entirely disappeared from the dairies of Europe, Britain, and America.

In this regard two very suggestive series of experiments are open to some one in the future:

1. Can or does vaccine acquire its original or gain in virulence by being transmitted from human being to human being through hundreds of generations?

(There are so many dangers to mankind from other diseases that it is doubtful if any one would be justified in entering upon such an experimental study.)

The other question has not these objections and it is a wonder that no one has entered upon it. It is:

2. Can we not prove that vaccine is but bovinated small-pox by inoculating cattle (heifers) from a small-pox patient, and then carry the inoculation through many generations of cattle and thus acquire, or prove, *vaccina* again?

To Jenner in the first place, and Pasteur in the second, the world principally owes the idea of inoculative prevention. It is easy to be seen that this idea is a development—a slow growth. Pasteur has endeavored to apply the principle to the prevention of several animal diseases. With regard to rabies it is my opinion that he has most signally failed, but his work in that direction has by no means failed in giving him the most sensational reputation of any man of the present day. In fact it has driven out of sight his far more instructive work upon anthrax and other diseases. While the results with Pasteur's vaccine against these diseases have not been generally accepted, particularly on account of the looseness with which Pasteur collects and publishes statistical results, still there is no question

whatever that they are worthy of credence, and that they can prevent the natural outbreak of the disease against which they are directed. It is somewhat singular, at first sight, that although the German government has given Pasteur several opportunities to demonstrate the preventive value of his vaccine against anthrax and rouget, and that although the results have been more or less favorable to Pasteur, and been repeated by German investigators, still their practical application has never been adopted by the government. There must be some reason for this, for that government not only has more competent workers than France at its command, but more numerous and better appointed laboratories. The reason for this is simple. The Germans saw immediately that more satisfactory, better, and safer results were to be obtained through the methods of isolation and separation of the well from the sick (in the same diseases) at the hands of their efficient veterinary police than by Pasteur's methods.

My skepticism as to the value of Pasteur's anti-rabie inoculation is based entirely on practical and scientific grounds. In fact, Pasteur has no rational fundament to stand upon in this disease. His position, in this regard, shows how essential a thorough medical education is to any one entering upon the field of prophylactic research. When one essays this work of protective, or preventive, inoculation, the first question to be settled, before any experimental work is begun, is, does the nature and character of the disease offer any phenomena which warrants the hypothesis that it is preventable by artificial inoculation? In other words, under natural conditions, do people or individuals often recover? Is the disease often characterized by mild attacks which confer an equally reliable immunity to that acquired by such as survive a severe attack?

These conditions are known to exist in small-pox, mumps, measles, and other contagious diseases; the late Professor Frank, of Munich, thought he saw evidence that they did in anthrax; we know they do in the swine plague, Rouget, hen cholera, yellow fever, the southern cattle plague, and other diseases. We do not know that they exist in Asiatic cholera, and every one knows there is not on record a single case of mild or recovered rabies, far less one showing that such recovery produced immunity towards a second infection.

We do not even know to what causes to attribute these mild attacks. We do not know whether they should be sought in certain idiosyn-

cratic powers of resistance in individuals, or in a prevailing mitigation of virulence in the specific etiological factor at one time which does not exist in another, nor have we any idea what influences have been at work causing such mitigation. That constitutional idiosyncracies play a very essential role is well illustrated by outbreaks of scarlet in a family of children at almost one and the same time; that there must be also peculiar circumstances, some genius epidemicus, which can and does act in mitigating the virulence of the specific cause is also seen in different outbreaks in different years of this same disease, for at some times it is very mild, and nearly every child attacked recovers, while the next year it may be a fiend incarnate in its murderous ravages.

Now none of these things exist in rabies. Given an actually rabid dog and persons bitten by the same, those in whom the disease occurs all have the same symptoms and all die.

Not one traveler on this terrible road of torture has ever recovered the path to health. All have gone to the place from which no sufferer has ever returned.

With these facts staring him in the face, what grounds had Pasteur for attempting to prevent such a disease by inoculation?

Without ever having produced one single case of rabies in a dog, and shown by the natural experiment of letting such a dog bite healthy ones, and then seeing rabies result, what right has Pasteur to say that he has ever caused actual rabies by inoculation?

When the specific cause of rabies is found, and actual rabies produced by inoculation of healthy dogs, and the same disease induced in other healthy dogs by bites from the former; when such bites are seen to induce mild cases which recover, and when such dogs can successfully withstand the bites of known rabid dogs, as also the bites of dogs made actually rabid by inoculation, and again the inoculation with the same material, and all this evidence is controlled by carefully watched and inoculated animals, then and only then dare Pasteur or any of his followers say that rabies is preventable by inoculation.

Notwithstanding this skepticism, the fact exists that inoculation will prevent to a certain degree the natural eruption of certain diseases. On the other hand, the German government is absolutely correct in placing the utmost value upon well-proven restrictive and hygienic measure, for have they not ever before them the historic fact

that the vigorous application of such measures has entirely freed their country from the two worst pestilences of past ages, the bubo pest and black death, and that the same treatment has kept them free from cholera in the last few years, and that the rinderpest no longer carries terror to the homes of the agriculturalists. Again, as has been said, although the civilized world is fully aware of the protective value of vaccination in small-pox, still we also know that its absolute execution among a people, especially the unfortunate and poorer classes, is a practical impossibility ; nor do we feel thoroughly certain that it can be relied upon to prevent, in every case, where it has been done, for many people do not repeat it often enough, and even the small-pox itself gives the exceptions, that only go to prove the rule that an occasional person may become infected a second time. Hence, every sensible and wide-awake community will never neglect to employ the most trustworthy hygienic and restrictive measures to protect itself, or its animal property from the ravages of diseases of a pestilential character. Another objection to the preventive inoculation of live-stock is, that it is very liable to render owners careless with regard to taking due precautions, or to repeating the inoculation, for it is admitted by Pasteur that the period is short for which such immunity is conferred in strictly infectious diseases. In this regard it will take many years of work and many workers to establish the period for which such inoculation has value. Contagious diseases, in which we have any reliable experience, such as small-pox and contagious pleuro-pneumonia, again differentiate themselves most strongly from infectious, as not only the survival of the natural disease, but artificial inoculation produces an immunity which can, generally, be trusted to continue for a term of years. Again, inoculation in contagious diseases gives the most positive evidence that such septicæmiæ as anthrax, Rouget, swine plague, hen cholera, etc., are really extra-organismal infections. No case is recorded where it can be clearly shown that a vaccinated human being, or an inoculated bovine, ever extended either the small-pox or pleuro-pneumonia to healthy individuals ! Quite the contrary is the case with regard to the extra-organismal infectious diseases, in which the sick or inoculated animal may be the means of infecting healthy animals by passing the inoculated germs off in its feces or urine, and again infecting land or earthy surroundings. That this has occurred in the "Rouget"

has already been shown by quotations from Lydtin and Schottelius. In this case the results of inoculation were so suspicious that, though no one could deny its efficacy, still the careful Germans have made no further use of it, preferring to rely on restrictive and hygienic measures for the protection of their swine. The same must also be the case in anthrax, for we know the germs are in the material of Pasteur's vaccine, and I have personally found the manure of cattle diseased with anthrax replete with germs immediately after being dropped. A vaccine prepared according to Pasteur's method would also have the same evil effects in swine plague. With this fact most painfully in mind, and realizing the immense responsibility towards our live-stock interests which are incumbent upon my position, I have endeavored to go very slowly and with the greatest caution in all my experimentation. Unfortunately the urgent demand that I publish what I had been doing, over a year ago, to satisfy the public, gave my enemies in Nebraska the opportunity to demand public tests, which, the more I thought of them, the more determined was I should not be given. The recklessness of these people towards the live-stock interests of the state can be at once appreciated. As will be shown, there is no question but what inoculation can be done and immunity to natural infection produced in swine, which will certainly continue for eighteen months or two years. But I positively assert *that it would be a malicious disregard of the public welfare to introduce this present method of inoculation indiscriminately, though it may be justifiable on farms that have become permanently infected.* Otherwise, every fact in the natural history of swine plague plainly shows that the indiscriminate inoculation with material prepared as it now is, would be only a means of extending the disease to places where it never had existed, and of reinfecting those already infected, and only lead to bringing disgrace upon myself and a possible means of prevention in the future into disrepute. The real malice in those who demanded inoculation tests was hidden behind apparent public interest. From my own writings they knew I would not be guilty of an act of inconsiderateness, but they hoped to stir up a public demand, and trusted the accidents which I had foreseen would soon lead to my expulsion from the state, and thus to the "stamping out" of myself as an investigator and the impossibility of the completion and publication of this report.

If I am right in my assertion that all these diseases, swine plague

included, are blood poisons, and there is no question on that point, then I am confident that human ingenuity and perseverance will eventually separate that poison from the microbaic poison producer, and I am equally confident that those same human agencies will eventually produce a safe material that can be depended upon to produce immunity by inoculation with absolute freedom of any danger of extending the disease to healthy stock, or of reinoculating or infecting the surroundings of such animals.

I am confident of this! Why should we be discouraged? By work which has not occupied six months' actual time we have completely laid the foundation and, I say it proudly but not boastfully, settled questions forever that the Agricultural Department at Washington has been vainly trying to solve ever since 1878. Had I the time, and were not this Bulletin already too extensive, I would give evidence showing that this problem has so much testimony on its side that its eventual, successful solution is only a matter of time and perseverance. The real solution belongs to the chemist. It is the province of the patho-bacteriologist, however, to carry the experimentation so far as to demonstrate that the chemist can do it. This is the very work I have essayed to do, and been needlessly obstructed in doing, ever since January 1, 1887. Not having the place and means, I gave my attention to investigations upon the southern cattle plague, the results of which will, it is hoped, sufficiently pardon my inability in the other direction.

Above I have said that our present method of inoculation may be, perhaps, justifiably practiced upon swine in permanently infected localities, and, as it has also been said that the disease is a septicæmia, I now desire to produce evidence where inoculation is and has been successfully practiced upon animals living in such permanently infected districts, and also a disease that is an extra-organismal infection, though caused by a different organism than the swine plague. This disease is the most inexplicable thief of all animal diseases, because it selects only the best stock, and the young among them. I have reference to the celebrated "Black Leg," or, as it is technically termed,

"EMPHYSEMA INFECTIONOSUM," SUCCESSFULLY PREVENTED BY
INOCULATION.

I quote from the work of my friend, Prof. Kitt, of the Munich Veterinary School, upon "Schutzimpfungen:—"

"The results of protective inoculation in Switzerland upon 1,810 head of cattle have been accurately ascertained. These 1,810 cattle grazed upon land that had become notoriously "Black leg" infested in the valleys of the Alps, and were kept upon these districts throughout an entire summer.

"Of these 1,810 inoculated cattle, only two became infected; one, four and a half months, the other two months after the completion of the inoculative treatment.

"Upon twenty-four Alps there was the proportion of 908 inoculated cattle to 1,650 non-inoculated, so that the two inoculated animals, which died, have reference to this 908, a loss of 0.22 per cent. Of the 1,650 uninoculated cattle, 101 died of the black leg, or a loss of 6.1 per cent."

These figures, says Strebel, speak most decidedly for the value of preventive inoculation in black leg, and cannot be attributed to any accidental influence. Kitt, who otherwise is a strong opponent of inoculation, says:

"The results are even more favorable than Strebel indicates when one considers the circumstances more critically." p. 146.

These results have been so repeatedly confirmed that the inoculation of young cattle, which are annually grazed upon these permanently infected mountainous valleys and slopes, has been made obligatory in the various Swiss cantons.

Here again caution is necessary!

The American stock raiser must remember that this procedure, as at present conducted, is only allowable where cattle continually graze upon such isolated and permanently infected districts as exist in the Swiss Alps, where these districts are well known, and where it is practically impossible for the cattle to wander about and thus infect districts that are known to be free from the disease.

As I have said, and as I wrote last fall, this fact of the danger of extending these local infectious, or extra-organismal, septicæmic diseases of our live-stock by the present method of preventive inoculation, has ever been before my mind, and my responsibility to the public instead of becoming less is continually augmenting. The very logical objections of trustworthy German investigators to this method of inoculation has not escaped my notice. It may be well that I state what this method is. It is that of Pasteur, by which, in one manner or another, specific micro-organisms are either robbed of their

power of producing as virulent (septic) poison, or else they produce it so slowly that the organism infected (inoculated) is able to adapt itself to it before it has time to exercise any fatal effects. The intra-organismal hardening thus produced continues for an unknown period. It is like learning to smoke by beginning with the mildest cigars attainable, and gradually passing to stronger as one gets accustomed to the influence of the tobacco. Now in this material the germs are always present. It is a well-known fact that Pasteur's vaccines cannot be depended upon for constancy of virulent action. The second or strong vaccine has often been found less active than the primary or weaker. Here is a very grave error, which has brought much discredit upon Pasteur. In fact all of his work partakes of this uncertainty. The cause is to be sought in the fact that having no idea of pathology, or disease, he is continually groping in the dark. Yet he is no less an important explorer in an unknown field. I long ago saw that the next important point to be gained, after demonstrating that immunity could be conferred by inoculation, according to Pasteur's methods, must be some means of acquiring an excessively virulent material of absolutely constant strength. Since January, 1887, I have waited for the means to do that with whatever patience I could command, and only recently could I make a single experiment in that direction. Just as I had evidence which largely indicated that I had the means of settling this, the next important step in the development of this question, I had to stop for want of the kind of animals necessary to the work. The reader must understand that the testing of such a question as this is by no means the simple matter it appears. Hundreds of experimental inoculations have to frequently be made upon all kinds of animals in order to find the species which will give the desired result. After using up every available animal, I had, as I said above, to stop for want of means. But stopping does not mean giving up!

The fact that Lydtin and Schottelius record positive evidence of the extension of Rouget to healthy hogs through those inoculated by Pasteur's agent, cries in a most startling manner, "Halt!" to that kind of work where it is done so indiscriminately as in France, and may be the ground which explains the continued increase of Rouget in France, or, perhaps more justly, the far greater percentage of that disease, and even anthrax in France in comparison with Germany, notwithstand-

ing the immense number of inoculations annually performed under Pasteur's method. In reality all the evidence of scientific experiment and observation certainly goes to show that instead of being a benefit to France, the present Pasteurism is a constant means of keeping up, and perhaps extending also, these extra-organismal septicæmiæ over the country

This possible and really probable danger of Pasteurism, as applied to preventive inoculation, simply condemns the method. It does not mitigate against the value of the principle, of which it is really a firm link in the chain of evidence which is surely to prove of incalculable benefit to mankind. The very difficulties and intricacies which are thus reached should only stimulate us to continued and more persistent endeavor.

Now I say that what man has done in one way can be done in another. If Panum, Bergmann, Brieger, and others have separated septic salts from one kind of putrid material; if Brieger can isolate tetanin from the tissues of a person dying from lockjaw; if other toxins have been thus obtained and specific symptoms produced by the injection of these, as solutions, into the blood of healthy individuals, then it can be done in regard to these extra-organismal, infectious septicæmiæ. We know that it is not the bacilli which directly cause the black leg, any more than it is the bacteria which directly cause swine plague; on the contrary, we know that in either case the bacteria produce the specific disease by the secretion of a specific sepsin, or poison, which is carried over the system by the blood, and thus causes those lesions which are specific to each disease. We know the specific lesions in these diseases are those of septicæmia. We know that the micro-organisms are essentially different in their physiological natures, as essentially as they are in form, because entirely different lesions are the result of the action of this septic solution in the blood in each disease.

Now this poison is chemical in nature, and it may be positively relied upon that some day, be it sooner or later, that poison will be isolated as a salt, capable of inducing the very septicæmia the micro-organisms do in producing the respective septicæmic diseases, and then, and only then, will an artificial means of prevention of such diseases be in the hands of our suffering live-stock breeders. Experience can only show whether it will prove practical or not. As I said before, the work of

preparing the way is in the hands of the patho-bacteriologist. Until he has built the road inch by inch, the chemist has nothing to do with the matter. If successful, they will solve a problem, in comparison with which the work of Jenner is but an infant, prodigious as it has been in its far-reaching blessings. But let us not belittle the world's gifted ones. Jenner showed the way, Pasteur built upon and enlarged it. America enjoys the benefits, having contributed nothing to aid in the work. Now that she has begun, let the good work proceed. Let Nebraska, that has really done more than any other English-speaking state, show that she values her own reputation, and that she can see it is her duty to support science for science's sake, even though victory tarry long.

With these introductory remarks, we may now turn to the

EVIDENCE WHICH SHOWS THAT INOCULATION MUST EVENTUALLY BE ACHIEVED IN SWINE PLAGUE.

When the pathological experimentalist approaches a question of this kind, the subject at once differentiates itself into two essentially different, yet very closely related, phases.

We have to establish two facts :

1. That hogs which have recovered from an attack of hog cholera are not, as a rule, liable to a second attack within a reasonable length of time.
2. That we can induce a mild attack of hog cholera by means of an artificially prepared cultivation of the germ of swine plague, when inoculated upon previously healthy hogs that have never had the disease.

Let us give our attention to the first condition.

Among the earliest evidence as to any peculiarities of swine plague which I gained from conversation with practical hog breeders in Nebraska, was the generally expressed opinion that hogs which had been through the swine plague and recovered seldom had a second attack.

This was not a new point to me by any means, as I knew the various writers upon the disease had all said the same thing. Now, when one finds every experienced hog raiser in a state as large as this is; when one corresponds with many others; when one attends conventions of these men, and finds no exception to the general testimony, save in degree; and when, beyond this, one meets and finally

becomes intimately acquainted with men noted for their money-making success as raisers of swine, and such men tell him *that they owe some of their success to the fact that they make it a point to buy every sow they can which has recovered from the swine plague*—I say, when one finds all this evidence on the positive, and only sufficient to endorse it on the negative, side of the question, *there is no man audacious enough, unless an ignorant and conceited ignoramus, to question the fact that swine plague is a non-recurrent disease.*

However much I may object, and I most seriously do contradict Mr. Salmon's definition of swine plague as a "non-recurrent fever," or a "non-recurrent contagious fever," there is no question whatever about the correctness of the general assertion that one attack, generally, produces secure immunity against a second during the period that swine are allowed to live.

Prof. Law, Dr. Detmers, and other observers confirm this opinion.

"All experienced feeders agree in the opinion that animals having the disease and recovering from it seldom have a second attack, and they state that in purchasing animals to feed preference is always given to those that have gone through with the disease." Dr. D. W. Volles, U. S. Ag. Report, 1883, p. 429.

"I feel quite satisfied that numbers of pigs pass through the disease without manifesting any recognizable external symptoms, no matter how carefully they may be watched.

"Does one attack protect against future attacks?"

"In the past few months I have had the opportunity of watching several animals that, during a previous epizootic, were attacked by this malady and recovered, and which during the present outbreak have successfully resisted its influence." Principal Walley, l. c.

That swine can become infected "and pass through the disease without manifesting any recognizable external symptoms," as Prof. Walley says, is most conclusively shown by my second series of inoculation experiments in 1887, and especially endorsed by the autopsy of a pig which was purposely killed as recorded in the necroscopical notes. See autopsy XXIII. Furthermore, I have tested this question by examinations in the field, and it has become more and more an acknowledged fact among swine breeders that have some interest in this work and have given much attention to the matter. There is a herd of swine belonging to Hon. S. W. Burnham, near Lincoln, that were raised upon permanently infected lands, every one of which has

shown an acquired immunity to swine plague, though three separate lots of young hogs that have been most carefully purchased have nearly all died off when put on the same land during the past three months. Were it necessary, this statement could be endorsed by numerous letters, but I will let it stand as it is, as a suggestion to practical swine breeders that careful watching of their own hogs will give the most practical evidence that preventive inoculation is surely to be the final result of determined investigation.

European observers have had so little actual experience with the swine plague that they still know very little about its deportment among swine, and do not really know what the disease is.

The next thing was to

PROVE THESE PRACTICAL OBSERVATIONS BY SEVERE EXPERIMENTS.

The following list represents the experiments done at this station prior to Jan. 1, 1887:

TABLE OF EXPERIMENTS.

1. Six-months-old hog sick with swine plague (recovered).
2. Six months old hog recovered from swine plague last winter (no effect).
3. Six-months-old hog sick with swine plague, died.
4. Six-months-old hog recovered from swine plague last winter, no effect.
5. Three-months-old hog in pen with No. 4 after 3 had died, no effect.
6. Three-months-old hog inoculated with culture from Mr. W.'s pig (of July 8, '86,) July 20, '86, died July 25, '86.
7. Three-months-old hog inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, died Aug. 1, '86.
8. Three-months-old hog inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, died Aug. 7, '86.
9. Three-months-old hog inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, recovered.
10. Same as No. 2, inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, no effect of consequence.
11. Same as No. 4, inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, no effect of consequence.

12. Same as No. 1, inoculated with culture from Mr. W.'s pig (of July 8, '86) July 20, '86, no effect of consequence.

13. Another pig, same as Nos. 2 and 4, inoculated with the same material, no effect of consequence.

14. Healthy pig inoculated with tenth generation of same material as 6 to 13, Sept. 28, died Oct. 13, '86.

15. Healthy pig inoculated with third generation of material from Rising, Neb., Sept. 28, died Oct. 7, '86.

16. Healthy pig inoculated with third generation of material from Rising, Neb., Sept. 28, recovered.

17. Healthy pig inoculated with third generation of material from Rising, Neb., Sept. 28, died Oct. 15.

18. Healthy pig inoculated with third generation of material from Rising, Neb., Sept. 28, died Oct. 16.

19. Healthy pig inoculated with third generation of material from Rising, Neb., Sept. 28, recovered.

20. Healthy pig fed with potato culture of same bacteria Oct 23, no ill effects.

21. Healthy pig inoculated with three grammes bouillon culture from spleen of sick pig Oct. 27, died Nov. 18, lesions very marked.

22. Same as No. 2	{ Inoculated with 5 fl. grammes of a pure bouillon culture from the Rising outbreak, which killed rabbits in four days. Some swelling and heat at point inoculated, and lameness. No other evil effects.
23. Same as No. 4	
24. Same as No. 13	
25. Same as No. 19	

26.	{ All raised at college farm from sows that had had the swine plague the previous winter and recovered. Each inoculated with three fluid grammes of a vaccine on the 14th of November, '86. Somewhat off feed for a few days. Heat and swelling at point inoculated. Again inoculated with a stronger material on Nov. 27, '86. Appetite not much affected. Some swelling and heat at points inoculated.
27.	
28.	
29.	
30.	
31.	
32.	

Healthy pigs 6 mos. old.

Let us analyze the above list a little for the convenience of the reader! Nos. 2 and 4 represent two hogs that had been through the

swine plague in the winter of 1885-6. Nos. 10 and 11 represent the same animals, while No. 12 is the same as No. 1, and is the recovered hog of the two very sick ones presented me by Mr. Wagner in June, 1886, mention of which is made in our neeroscopic notes in describing the lesions observed in its companion.

The tests these three animals were put to should be seen to be appreciated, for it is well known that the writer does not mean any "child's play" when he undertakes anything. In fact, I can truly say, that in every test which I have made I have invariably meant to kill if such a thing was possible when either exposure to natural infection or inoculated material has been used, either to test these recovered hogs or those which had been subjected to preventive inoculation.

It will be observed that the Nos. 1 and 2 were penned together. No. 1 was a very sick hog. They staid together till No. 1 was fully recovered. No. 3 penned with No. 4 represents the Wagner hog that died. After that these hogs, 1, 2, and 4, were repeatedly inoculated with large amounts of material, the quality of which was tested on healthy pigs, but they were never made sick thereby. I will here mention that these three hogs, with two other recovered ones belonging to the same lot as Nos. 1 and 2, and some of the inoculated hogs to which reference will soon be made, were subjected to six fluid grammes of an excessively virulent material injected directly into the abdomen or into the flank, in the summer of 1887. If the expression may be allowed, the results fairly made them tremble. Only one died, and the lesions resulting have been described in the autopsy of pig, 45, No. XXXI. Such lesions have never been recorded in a case of natural infection. Cuts representing these intestinal lesions may be seen, Plates VII. and VIII. The others finally recovered. The severe tests made in 1886 most conclusively show the nature of the immunity in swine recovering from natural infection.

Hogs which had the swine plague could not be reinfected by inoculation, nor by contact, nor by pen infection, as they were not only placed in the pens with sick hogs, but also in the same pens with the pigs killed by inoculation with pure virus.

The next question was to expose these hogs to natural infection in the most extreme manner possible. A most severe outbreak of swine plague at the farm of a Mr. B., near Lincoln, offered not only a fitting opportunity, but the severe snow-storm of November 17th, in which

my test hogs were snowed in with a large number of sick, and a dozen or more dead hogs, added to the severity of the test. All they had to eat for three days was the cadavers of their dead companions.

EXPERIMENT.

Five hogs that had had swine plague the previous winter at the college farm, four of which had been repeatedly tested as mentioned above, and one which had not been so tested, as well as Nos. 26 to 32 inclusive, and two non-treated pigs (control pigs), were sent to the place mentioned.

The results of this experiment were not as satisfactory as could be wished, for various reasons.

First—The smaller pigs were rather fine quality, and four of them were either stolen or run over by the cars which went close by the field where the sick drove was, and the wire fence did not keep the smaller ones in.

Second—The two control pigs and one vaccinated pig died of the swine plague or the storm, but had the lesions of swine plague; they were found covered with snow and frozen stiff, so that an autopsy was only made at some inconvenience. Cultures grew from the spleen, and mice succumbed to the cultures.

Two of the inoculated pigs, being small and somewhat weak, died the second day after their return to the college farm, from general exhaustion. No lesions of consequence; nor were bacteria to be found in them either by examination of their blood with the microscope or by cultivations.

Fourth—The hogs from the college farm that had the swine plague, and been already severely tested, as well as the one from the same lot that had not been so tested, went through this exposure to infection and storm without any visible ill effects, though exposed for twenty-three days, and have remained well ever since.

The result obtained then had only this value: It completely demonstrated that the majority of hogs that have been through swine plague from natural infection will not have the disease a second time, even after very severe tests, and therefore that

Prevention by inoculation is possible.

I also inoculated some forty of Mr. Burnham's hogs, and very severely; these hogs came through and thrived better than any other

hogs on the place. Whether this fact was indeed due to the inoculation or not cannot be positively asserted, but there is a favorable suspicion that it was.

I wish to publicly express my thanks to Mr. Burnham for the extremely kind and liberal support he gave me in my work, placing, as he did, his man and everything I desired at my disposal.

TEST EXPERIMENTS.

The next thing was to try the vaccine test over again. To this end fresh pigs numbered with tags in the ears were selected; their numbers were 41, 42, 43, 44, 45, 46, 47. Nos. 42, 43, 44, 45, 46, 47 were first inoculated with four fluid grammes—in the inside of the hind leg—of a mild vaccine, upon November 4, 1886, and again with six fluid grammes of a stronger vaccine upon November 27th. While thrown off their feed, and somewhat lame, with some heat and swelling in the inside of the hind leg, they did not seem to suffer very much. No. 41, which was the same pig as number 20 in the previous list that had been fed with pure potato cultivations, was inoculated with the same amount of vaccine as the others (six fluid grammes), on the 27th of November.

December 27th, Nos. 41 and 44 were inoculated with six fluid grammes of a fresh culture (second generation) made from the spleen of a hog from a very virulent outbreak at Valparaiso, Neb. These hogs were inoculated directly into the abdominal cavity. On the same day and at the same time Nos. 47 and 46 were inoculated with the same material in the inside of the hind leg.

December 28th, No. 41 died from the effects of the bacteria only (see Autopsy No. XXII.), that is of septicæmia, or blood poison, as can be seen from autopsy No. I. Its blood and the tissues of the spleen were literally swarming with the well-known bacteria. Though frozen solid, cultivations grew from the spleen, and two rats inoculated succumbed in forty-eight hours. Pure cultivations were obtained from the rat, and have since been retested and again proved fatal.

Nos. 44, 42, and 46, that had been vaccinated twice in thirty days, were somewhat off their feed and lame a few days, but entirely recovered, while 41, that was only inoculated once, succumbed.

On December 12th, numbers 45 and 47 were again inoculated with six fluid grammes of a bouillon culture that had previously been tested

upon rats and proved fatal. These pigs also recovered completely, and may be considered proof against swine plague.

We have therefore positively proved that we can prevent swine plague by inoculation of healthy swine with an artificially prepared virus.

The results of these experiments have been called in question by a state official in Nebraska.

Now, I say again, that severer tests could not possibly be made and there are too many of them and they are of too positive a nature to be negatived by any amount of contrary testimony.

First let me give an example of the educational and professional qualifications of this interfering obstructionist.

“To the farmers and stock growers of Nebraska—In accordance with a resolution of the live-stock, sanitary commission of Nebraska, adopted November 29, 1887, the following circular has been prepared by the state veterinarian for distribution. The object of this circular is to enlighten farmers and stock raisers of this state.”

The kind of enlightenment given can be judged by the following :

“To disinfect harness, robes, grain-bags, blankets, and all textile fabrics, including ropes used about diseased or suspected animals, boil in a solution of carbolic acid (two ounces of the acid to one gallon of water), or fumigate with sulphur as described above.”

“Boil harnesses and robes” ! One would think the learned gentleman was a butcher and accustomed to scalding hogs in order to remove the bristles ! “Two ounces of carbolic acid to a gallon of water” ! Sixty parts acid to 4,000 water ! How the glanders bacilli must have enjoyed such a bath !

MR. SALMON ON THE PREVENTION OF SWINE PLAGUE BY INOCULATION.

Upon this subject he says :

“Our investigations have shown that the plague is a non-recurrent fever, and that the germs might be cultivated; they have even proved that these germs may be made to lose their virulent qualities and produce a mild affection. Surely we have here sufficient evidence to show that a reliable vaccine might be easily prepared, if we carried our investigations but a little way further. (!!!)

“If we had such a vaccine, if it were furnished in sufficient quantities

and of a reliable strength, if it proved safe in the hands of the farmer, would not our problem be solved?

"Could we reasonably expect anything more or better for this disease?" U. S. Ag. Report, 1883, p. 57.

The farmers who read that, the poor hog raiser and the great cattle and hog feeders of the West, must have felt a ray of hope dart through their breasts when they reflected upon the above very positive announcement of the ability of Mr. Salmon to send the oil of salvation over the country, and thus put an end to the annual decimation of their porcine herds, if Mr. Salmon only "carried his investigations but a little further."

This hopeful feeling on the part of the suffering hog raisers of the United States must have even sprung into a genuine flame when they found that so mighty an authority as "M. Pasteur had recently confirmed our (Salmon's) American investigations in a very complete manner. He shows that the disease is produced by a micrococcus, that it is non-recurrent, that the virus may be attenuated and protect from subsequent attacks." l. c.

Alas for human frailty! Alas for the shattered hopes of the farmer!

Where was the frailty? Who shattered the hopes of the farmer?

No less an authority than Mr. Salmon, who had so positively told them "that a reliable vaccine might be easily prepared if we carried our investigations but a little way further."

Why did he not keep this positive promise so "easily" made?

We have already shown the reason!

Because a "micrococcus" is not and never has been the cause of swine plague in the United States! Hence, the above assertion was made entirely out of whole cloth, for the reason that Pasteur had really given grounds (he kept his promise!) that a "vaccine contra Rouget" could be made, and M. Pasteur had said that a "micrococcus" was the cause of "Rouget," and everybody then thought that "Rouget" and swine plague were identical diseases, and so it was easy to make a "micrococcus" the cause of swine plague. Mr. Salmon is very fond of authoritative support, and to tell our suffering farmers "that a reliable vaccine might easily be prepared, if we carried our investigations but a little way further," was an apparently harmless piece of encouragement under the circumstances. But this whole fabric was doomed to a terrible collapse, and with it went the

Pasteur-Salmon micrococcus to the bourne from which no micrococcus, even, can ever return.

It suffered death! It went into "innocuous desuetude"!

The cruel monster to thus shatter the sacred authority was Prof. Schütz, of Berlin!

We have previously shown how he demonstrated that a "micrococcus" was not the cause of "Rouget," and that a bacillus was; but while that demonstration shattered the micrococcus of M. Pasteur, it had no necessarily evil effect upon that of Mr. Salmon, had it really existed as the cause of swine plague in the United States.

But Mr. Salmon did not wait to see whether it really existed or not, or whether it had only taken a quiet rest in "innocuous desuetude"?

Here was a new authority, but it would not do to follow it exactly, so Mr. Salmon drops his "micrococcus," very gently, by saying that "we no longer consider that all outbreaks of swine plague are due to a micrococcus."

That is what Mr. Salmon told the swine breeders in his next report, 1885.

Even under the above language, the "outbreaks of swine plague" that were due to that "micrococcus" should have still been preventable by that "reliable vaccine," but even for these few we have never heard that those "investigations" have been "carried a little way further," and we know that in his conclusions as to his "new microbe" Mr. Salmon entirely gave up any etiological connection of his "micrococcus" with swine plague, for has he not told us that "the preceding investigations definitely settle certain controverted points in the etiology of swine plague"? 1885, l. c., 229.

Such as:

"Swine plague is caused by a specific microbe multiplying in the body of the diseased animal. This microbe belongs to the genus bacterium." p. 229.

It will be remembered that in the same report Mr. Salmon had already said, "that we no longer consider a micrococcus to be the cause of all outbreaks of swine plague" (p. 186), but if we turn over a few pages we shall see evidence that no outbreaks of swine plague were caused by this "micrococcus." In fact, we shall find that, notwithstanding all Mr. Salmon had said of that "micrococcus" in all his

previous reports, that until now he had never seen the germ of swine plague, for he tells us :

"The earlier work recorded in the preceding pages aimed to determine what relation the bacillus cultivated by Pasteur as a vaccine for Rouget bore to the disease among swine prevailing in this country." 1885, p. 217.

What a convenient thing a poor memory is !

How does the above remark compare with the passage previously quoted, where Mr. Salmon says :

"M. Pasteur has confirmed our American investigations in a very complete manner. He shows that the disease is produced by a micrococcus." ! l. c.

Let us now continue with the report of 1885 ! Immediately following the words previously quoted, Mr. Salmon continues :

"At that time the bacterium of swine plague had not been seen by us." l. c.

Can any one ask for more conclusive evidence that Mr. Salmon's "micrococcus" (1880-1885) never had any connection with swine plague? Can any one comprehend what could have induced Mr. Salmon to say (of the true swine plague germ, but which he falsely asserts to be the cause of a "chronic pneumonia," Report, 1886), "it is probably identical with the micrococcus described in my report of 1884,"* when he has said a year after that (1885) "that the bacterium of swine plague had not been seen by us," and again, "the bacterium which we have lately discovered, and which we believe to be the cause of swine plague"? l. c., p. 219. That last quotation settles the conundrum how the swine plague germ of 1885 became the "hog cholera microbe" of subsequent years !

It must have been through faith? Mr. Salmon only believed that his "investigations" (of 1885) "definitely settled certain controverted points in the etiology of swine plague." l. c.

In such an erratic personage it would be quite hard to say, at this moment, what he "believe(s) to be the cause of swine plague," "hog cholera," or his "chronic pneumonia" of 1886, which, according to his belief has become a very acute attack since then?

The reader will remember that, in 1883, Mr. Salmon asserted of his "micrococcus," "that surely we have sufficient evidence to show

* Journal Com. Med., l. c., p. 142.

that a reliable vaccine might easily be prepared, if we carried our (his) investigations a little way further." l. c.

Notwithstanding he gave up his micrococcus in 1885, Mr. Salmon "carried his investigations a little way further" into this question of the prevention of swine plague with "the bacterium which we have lately discovered, and which, we believe to be the cause of swine plague."

This time he took to pigeons, but he gives us equally reliable evidence "to decide a scientific question of this kind." This evidence, like all his former testimony, is the result of experiment. As in former cases, Mr. Salmon is very positive! He does not even believe! He knows it! He says:

"There can be doubt, therefore, from this very positive result, that the pigeons had acquired an immunity through the effect upon the tissues of the chemical products of the bacterium—of swine plague—in the culture liquid.

"The conclusions to be drawn, we believe (!), are of a superlative importance to a correct understanding of the phenomena of contagious diseases, and the methods by which these diseases are to be combated. They probably (!) apply to all bacterial plagues of man and animals in which one attack confers immunity from the effects of that particular virus in the future."

According to Mr. Salmon, they should apply to the swine plague, for has he not told us that it is a "non-recurrent fever." Next he confirms his belief by conclusions, as follows:

"1. Immunity is the result of the exposure of the bioplasm of the animal body to the chemical products of the growth of the specific microbe which constitutes the virus of contagious fevers.

"2. These particular chemical products are produced by the growth of the microbe in suitable culture liquids in the laboratory, as well as in the liquids and tissues of the body.

"3. Immunity may be produced by introducing into the animal body such chemical products that have been produced in the laboratory." p. 221.

That was in 1885! Why did not Mr. Salmon carry "investigations a little way further," which promised so much, into swine, and thus keep his promise of 1883, even if it was a little late in the day?

So plausible have the results of the experiments upon which the above conclusions are based appeared to Hueppe, that he has endorsed

Mr. Salmon and given him credit for a great piece of investigation, as follows:

“On the 20th of February, 1886, Salmon and Smith gave positive experiments showing that protective immunity could be conferred by the inoculation of soluble substances. These experiments were made upon pigeons which became infected by the inoculation of the bacteria of hog cholera in the same manner as with those of the hen cholera and the ‘Wild or Schweineseuche.’” *Fortschritte d. Medicin*, Vol. 8, p. 292.

Hueppe seems to be absolutely unaware of the fact that the experiments he refers to were published in Mr. Salmon’s report of 1885, when he said that the peculiar organism, which he then called “the new microbe of swine plague,” was entirely different from that of Schütz. Hueppe is also unaware of the fact that that same organism suddenly became the cause of Mr. Salmon’s “hog cholera” in 1886, as he is that that same organism has no existence in the American swine plague.

Hueppe also does not know that Mr. Salmon also says of that same organism:

“Fowls have resisted all our inoculations with the hog cholera microbes.” *Journal of Comp. Med.*, l. e., p. 143.

This should apply to pigeons!

Then again I would call Hueppe’s attention to the two following quotations:

1. “The germ of hog cholera produces fatal effects when inoculated in mice, rabbits, guinea-pigs, pigeons, AND PIGS.” l. e., 140.

2. “We soon found that there was no indication for attenuating the virus (to confer immunity by inoculation), because the strongest virus might be introduced hypodermically with impunity.” l. e., 148.

How about those “fatal effects?”

How about that immunity produced in pigeons with the soluble substance given off by these same swine plague, hog cholera peculiarities in 1885?

Although it was in 1885 that Mr. Salmon first published the experiments to which Hueppe refers so flatteringly, the latter must have been utterly unaware that the same crude gentleman had completely negated them in his report of 1886, and also in still later publications, as will be shown.

The reader must not forget that the bacterium of 1885, which "definitely settled certain controverted points in the etiology of swine plague," suddenly became the "bacterium of hog cholera" in 1886, and that, although in 1885 it "caused intestinal lesions in chronic swine plague," in 1886-7-8 it "produced the most remarkable and extensive ulceration of the intestines," and that "in hog cholera it is the intestines" which are diseased, although Mr. Salmon does tell us with the greatest gravity that, "the severest lesions were in the lungs" (1886, p. 650), and that "the lungs were adherent in places" (i. e., 651). The above contradictions are but a fitting prelude to the abruptness with which Mr. Salmon knocks all his glorious promises of conferring immunity in the head, in this report. He does it as follows:

"The foregoing experiments demonstrate the important fact that pigs cannot be made insusceptible to hog cholera by subcutaneous injections of pure cultures of hog cholera bacteria. The experiments have been sufficiently varied to leave no doubt upon this point." p. 653.

The observing reader will see that when Mr. Salmon claimed to have produced immunity in pigeons, in 1885, with a material holding the septic, poisonous product (of his then swine plague germ) in solution, that had that organism really been the true cause of swine plague, as he said, Mr. Salmon had really solved the momentous question of the inoculative prevention of swine plague, and truly deserved all and more credit than Hueppe has given him for being the first investigator to really demonstrate that this far-reaching procedure could really be done.

Now, where I can, I desire to do as much justice to Mr. Salmon as to any one, for facts will prevail, eventually, against all and every authority.

It will be remembered that Dr. Detmers obtained possession of this so-called "hog cholera microbe" of Mr. Salmon's from Professor Persh, of Philadelphia, and while he says it is not the germ of swine plague, does show, by experiment, that it could and did produce septicæmia; hence, if Mr. Salmon really did make these experiments with his forged germ of hog cholera, and did produce immunity in pigeons, as he claims, he shows that the same can be done with the true germ of swine plague, as that disease is also a septicæmia, though Mr. Salmon denies it.

With this, apparently, positive evidence in his hands, why did not Mr. Salmon proceed with such valuable work in 1886 and 1887, not to speak of the present year?

Why does he not mention any further experiments of the same kind in his report of 1886, or in his later publications?

What is he employed for but to do this thing?

The true answer is easy to discover!

Because, Mr. Salmon is fully aware that no such organism as his swine plague "new microbe" of 1885, or his "hog cholera microbe" of 1886, '87, and '88, exists as an etiological moment in swine plague?

Because, had he done this, he knew it would have to be done with the genuine organism, and that would have been admitting the fact that his swine plague organism of 1885 had no existence in that disease, and also that all his work was erroneous and a mass of "unfounded statements." He would then have to admit that the work done in Nebraska, and by Detmers also, was correct, and that, he knew, would result in showing the American people how they had been imposed upon by him for this long term of years.

No better evidence of the truth of all my assertions could be asked than this!

I do not know what may be the conclusions of the reader, but I have no doubt that the "evidence furnished is all that could be required to decide a scientific question of this kind," and the kind is, that Mr. Salmon has utterly contradicted himself in every particular.

MORE EXPERIMENTAL PROOF THAT INOCULATION WILL PREVENT SWINE PLAGUE.

My previous experiments in the fall of 1886 were sufficient evidence, to me, that swine plague could be prevented by inoculation. There was one objection to them, however. The farmer could not exactly comprehend the value of experiments so exclusively scientific in character. The farmer always wants the eminently practical, something he is accustomed to, in order to be convinced. Hence, it was necessary to inoculate a number of pigs and then subject them to the test of actual exposure in a very severe outbreak of disease under natural conditions.

In my former experiments, the one question to be determined was,

could artificial immunity be produced by inoculation, utterly regardless of the after effect upon the development of the animals?

As was to be expected, the extra severe treatment to which the animals inoculated in 1886 were subjected stunted them in growth.

Hence, in 1887, I determined to go to the other extreme, and see how weak an inoculation would give satisfactory results. In order to get the necessary animals I had to wait until they were born and old enough.

On August 25, 1887, twenty pigs, three months old, were inoculated in the thin skin of the flank with 1^{ccm} of a weak virus.

The animals were not disturbed thereby. In fact they showed no ill-effects whatever. It excited comment from my friend, Mr. Courtney, superintendent of the "College Farm," (whom, by the way, I wish to thank publicly for his continued assistance and watchful kindness) so that I determined to sacrifice one of the pigs. The autopsy will be found in the appropriate place as number XXIII. That a mild yet significant case of swine plague resulted can easily be seen.

On September 9th, 1887, these pigs were again inoculated with 1^{ccm} of a stronger virus, into the abdominal cavity. No evil results! *Time has proven that the animals have not been stunted.*

I wish to say just here, that I frequently mentioned to Mr. Courtney "that I would not warrant one of those pigs to stand any severe test."

That we were both destined to be agreeably disappointed, the future remarks will show.

On November 27th, the remaining pigs, except four sows, were sent to my friend, Hon. S. W. Burnham, who again had a very mild outbreak of swine plague among his hogs. The chances of infection were small, as his hogs had the run of extremely large cattle feeding yards. I will close this side of the story by saying that no evil results occurred to the hogs at Mr. Burnham's.

This test not being severe enough, it fortunately happened that one did occur which was severe enough to answer the most exacting skepticism.

I will let a Mr. Wing, late of the Agricultural School, tell his side of the story, as the thoroughbred hogs in which it occurred were directly under his supervision:

“LINCOLN, NEB., Feb. 18, 1888.

“*Dr. F. S. Billings:*

MY DEAR SIR—Agreeably to your request of Feb. 13, I give herewith an account of the outbreak of hog cholera upon the college farm.

“In the fall of 1884, November or December, occurred the first outbreak. It raged with more or less violence until about Feb. 1, 1885, at which time all but about ten of the hogs on the place had died. The hogs at first were in the hog-house and pens adjoining, but were removed to the cattle yards, some dying in both places. In the spring they were all moved back again to the hog-house. In April, a thoroughbred sow was bought and placed in the pens where hogs had died during the winter. She died May 4, 1885, and none died after that.

“In December, 1887, some pigs born in August, descendants of the survivors of the outbreak of 1884-5, commenced to die. They were in the pens about the hog-house, and, with their ancestors, had been kept there constantly since the former outbreak. Twenty-two in all died on the following dates :

“Dec. 7, one ; Dec. 8, five ; Dec. 14, one ; Dec. 15, one ; Dec. 16, one ; Dec. 20, one ; Dec. 21, two ; Dec. 22, one ; Dec. 23, one ; Dec. 25, one ; Dec. 27, one ; Dec. 28, two ; in Dec. on unknown dates, two ; Jan. 21, one ; Jan. 26, one. Eighteen survive, 10 of which have been constantly in the infected pens. On Dec. 8, there were put in one of the pens where the disease was raging most violently, four inoculated pigs (the sows) and three uninoculated pigs [control animals] that had had no previous communication with the sick ones. A few days later six more inoculated pigs were added (these last had been taken to Mr. Burnham's). Of these, two inoculated pigs died on unknown dates subsequent to January 26, the three uninoculated ones died on unknown dates previous to January 21.

“I am, very truly,

“H. H. WING.”

I can complete some of the facts that have been left uncertain by this Mr. Wing. It is very singular that he should know the dates upon which most of his hogs died, and not of mine which were in the same pen. This is a fair sample of the assistance I have had in my work. Two of the control pigs put in December 8, died on the 18th and 23d of December, respectively. The third died later. I made sufficient necroscopical observations on the first two, though frozen stiff, to assure myself of the nature of their complaint.

Now what does this outbreak show ?

1st. That Mr. Salmon's “experience,” that the disease is extinct in winter, is mistaken, for here we had winter in optima forma.

On the question of the effects of cold, Prof. James Law makes the following interesting communication:

"A healthy pig was placed in a pen from which a sick one had been removed thirteen days previous. The pen had been swept out, but subjected to no disinfection other than the free circulation of air, and as the pig was placed in the pen on December 19, all moist objects had been frozen during the time the apartment had stood empty. The pig died on the fifteenth day, and the *post-mortem* lesions showed the operation of the poison. This case was an example of the rapidity of the fatal action of the poison, the lungs and brain being decidedly affected. It sufficiently demonstrates the preservation of the poison in covered buildings at a temperature below the freezing point." U. S. Ag. Report, 1883, p. 408.

2d. That two inoculated pigs succumbed, is but the exception which proves the rule, for it will be seen by comparing the date, "subsequent to January 26th," given by Wing, with those upon which the two healthy control pigs died, December 18th and 23d, that the inoculation did have some effect in enabling them to resist infection. The extremely weak character of the virus used to inoculate these animals must also be borne in mind. The other inoculated animals are still alive and thrifty.

Now here we have a test of the inoculative treatment of six pigs inoculated in 1886, and subjected to the severest possible artificial tests, with no loss. Nineteen pigs inoculated in 1887, ten of which suffered exposure in the most malignant outbreak of swine plague I have seen, with but a loss of two. The history and autopsy of the "college farm" hog which died December 12th, 1887, will convey some idea of the character of this outbreak.

Can more conclusive proof be desired that inoculation will prevent swine plague?

Though I do not intend to refer to the literature of this subject at this time, it would be unjust and discourteous not to briefly notice the remarks of two well-known, early experimenters upon the subject of inoculation.

Dr. Klein says:

"Quite recently I have ascertained that pigs inoculated with artificial cultures of these rods (started from the pig, mouse, or rabbit dead of the plague), or with the diseased organs of a mouse or rabbit, suffer from a mild form of the disease, which after one or two

weeks passes off completely. I have had pigs that had been twice inoculated, the first time with artificial cultures, the second time with diseased organs of a mouse or rabbit, and each time the pigs suffered from a mild form of the disease. They were then inoculated a third time with the juice of the diseased (fresh) lung of a pig dead of the plague; but, after a few days, to a week, they completely recovered. If healthy pigs (that have not previously been inoculated) are inoculated with material from the diseased (fresh) lung of a dead pig, they usually die from the plague in a virulent form. But in the above case they were protected by previous inoculations, not altogether against a new attack, but against a fatal attack." *Micro-organisms and Disease*, p. 98, 1885.

Prof. James Law, who was the first to experimentally demonstrate that swine plague could be prevented by inoculation, says :

"These experiments point to the most important conclusion that the poison of swine plague, when passed through the system of sheep or rats, becomes lessened in virulence, and usually conveys the disease back to the pig in a non-fatal form. Should this be sustained by further experiment, and should this, like some other bacteridian diseases, so affect the system that a second attack is rendered much milder or entirely prevented, it will open the way for a system of vicarious inoculation that will save our swine breeders from the excessive losses that now threaten the existence of this industry." *U. S. Ag. Report*, 1879, p. 426.

"While, therefore, it cannot be confidently affirmed that we can, at will, induce a mild form of this affection which shall protect the porcine system against a severe one, we have, in our experiments, a sufficient warrant and inducement to carry this experimental investigation to a certain and reliable conclusion. It remains for the experimental pathologist to determine the exact conditions under which such immunity can be acquired, and how long protection to the system is vouchsafed." *Ibid.*, 427-8.

I think I can justly claim that these early observations of Professor Law and Dr. Klein have received their fullest confirmation through the experiments conducted at this station.

As soon as the means will allow, this work will again be taken up with renewed vigor; but, as has been shown, no general use of this measure can be resorted to until it has been shown that immunity can be produced by inoculating with the pure chemical products of the bacteria. This done, the future and permanent success of preventive inoculation for swine plague, or other such disease, must pass into the

hands of the chemist. Until then the farmers of the West must rely upon the restrictive and hygienic measures so fully detailed in previous pages, and I can assure them that diligence and carefulness, with rigid isolation of the well from diseased animals, and the avoidance of places where sick hogs are or have been, will almost as completely check the ravages of swine plague as any inoculative treatment, though at the cost of more care and labor to the farmer.

My task is done for the present. It has been my earnest endeavor to place this whole question in all its completeness before the American public and the scientific world. It is time that the exact facts be known. If I have one desire, beyond any other, it is that the world finally, though too tardily, give due credit to Dr. H. J. Detmers for the first real discovery of the micro-etiological organism of swine plague. I have endeavored to do Dr. Klein justice also, and although he undoubtedly saw the organism before Dr. Detmers, I think my confrères will agree with me that his likening it to "*Bacillus subtilis*" is such an erroneous conception that the full credit of original discovery belongs to Dr. Detmers. With regard to Mr. Salmon's work, the facts are now honestly before the world for the first time. I challenge the most rigid comparison of the whole, as here presented, with the original. Of it I say, as of my own, "truth is the mightiest and will live forever," let us both stand upon our own merits, and justice will eventually be given to those who serve their country and the world faithfully. Demanding criticism in the same manner I have meted it out to others, I remain one whose only ambition is to live and die in honest service of the live-stock interests of my country and the public health of the world.

THE AUTHOR.

Lincoln, Neb., June 30, 1888.

ILLUSTRATIONS.

It is not only a duty but a pleasure for me to publicly express the great obligations I am under to the State Journal Publishing Company for their unusual interest and friendly assistance in publishing this work, but equally to Mr. Grupe, the accomplished wood engraver of this company, for his perseverance in executing the cuts herein used, and his obliging attention in endeavoring to carry out the details in a work which he had never before undertaken. The cuts of the lesions of swine plague which have been selected for illustrations have not been made from fresh material, but from specimens preserved in alcohol and selected for the purpose during the progress of the work.

FRONTISPIECE.

This represents a view of a portion of the work room of the laboratory.

PLATE I.

represents the various forms of micro-organismal life which have been represented to be the cause of swine plague by Mr. Salmon during the period, 1878 to date, in which he has been engaged in investigating swine plague in this country. It will be observed that no two of these organisms are alike. The reader will remember that Mr. Salmon claims, or has endeavored to claim, that his micrococcus of 1884—Fig. 2—is identical with the germ of swine plague of Detmers and myself, though he does not mention Dr. Detmers' name in that connection. In Fig. 5, Mr. Salmon illustrates this germ of swine plague, and I beg of the reader to compare it with Fig. 2, and see if he can find any resemblance between the two objects. The reader can then compare Mr. Salmon's Fig. 5 with Figs. 2, 3, and 4, Plate III., and he will see the resemblance between the four illustrations. In Fig. 3, Plate I., we have Mr. Salmon's illustration of his forged germ of swine plague as he illustrated it in his report of 1885,

while in Fig. 4 we have an illustration of the same object when it became the germ of hog cholera in the report of 1886. The increasing coloring of the pole-ends, and its approaching resemblance to his Fig. 5, is easily to be seen, while its departure from the original illustration, when the "new swine plague microbe"—1885—is also equally perceptible. Mr. Salmon has failed to illustrate his third germ of swine plague—Dr. Rose's germ from Nebraska.

PLATE II.

Here we have in Figs. 1 and 2 Dr. Klein's illustrations of bacillus subtilis, and in Figs. 3 and 4 his "bacillus of swine plague." That the latter have no resemblance to the former is beyond all question, but that he illustrates the germ of swine plague is to be easily seen by glancing at Fig. 5, which represents the morphological appearances of a hanging drop culture of the bacterium of swine plague.

PLATE III.

In Fig. 1 we have the original illustration of Dr. Detmers to which he so touchingly alludes in his letter when describing the many difficulties under which he worked in the earlier days of his investigations. In Fig. 2, however, we have an exact copy from one of Dr. Detmers' photographs. Any one can see that it has relation to the same organism and is the same microbe pictured in my own illustrations, Figs. 3 and 4. Fig. 5 represents the bacterium of rabbit septiciemia according to Baumgarten; and Fig. 6, the bacillus of Rothlauf according to Schütz.

The remainder of the illustrations need no particular mention, as the necessary remarks will be found in the text, or attached to the plates.

INDEX.

	PAGE
Agriculture, State Board of Nebraska.....	353
Anthrax.....	177, 188, 198, 272, 294, 373, 374
Appeal for a national laboratory, ..	349
Aristotle.....	11, 263
Aspiration of germs in dust of pens a cause in swine plague.....	24, 143
Authority not always authoritative.....	270, 285
Authoritative evidence that swine plague is a septicæmia.....	286
Autopsy No. 1.....	120
No. 2.....	123
No. 3.....	129
Nos. 4, 5, 6, 7.....	133
No. 8.....	134
No. 9.....	135
No. 10.....	139
Nos. 11, 12.....	140
No. 13.....	142
Nos. 14, 15.....	145
No. 16.....	258
No. 16a.....	308
No. 17.....	146
No. 18.....	147
Nos. 19, 20.....	148
No. 21.....	150
No. 22.....	154
No. 23.....	156
No. 24.....	157
No. 25.....	192
No. 26.....	194
No. 27.....	196
No. 28.....	310
No. 29.....	313
No. 30.....	158
No. 31.....	160
Axe, Prof.....	11
Bacillus suis—Detmers.....	40
Bacilli in swine plague.....	33, 40, 41, 45, 46, 47, 51, 61, 94, 165, 318, 401
Bacillus subtilis—Klein.....	165, 169, 318
Bacillus—Reeves.....	318
Bacterium of swine plague—Salmon—1885, of hog cholera—Salmon—1886.....	53, 55
57, 60, 62, 63, 64, 70, 73, 76, 247	
Genuine, of swine plague.....	70, 82, 93, 97, 102, 108

	PAGE
Bacteria in swine plague, Action of.....	282, 300, 302, 322
Bacilli in Rouget.....	44
Banham, Geo. A.....	165
Bang, Professor.....	11, 190, 217
Bichat.....	264
Black leg.....	378
Bollinger, Prof., on the "Wild-seuche".....	9, 188, 302, 338
Bowhill, Dr. Thomas.....	95, 166
Boerhaave.....	264
Brown, Prof., History of swine plague in England.....	9
Brownian movement.....	116
Breeder's Gazette.....	29, 63, 81
Budd, Dr. Wm.....	10
Burial of dead swine.....	368
Carsten-Harms.....	10
Cars as a means of extension. ...	32
Carriers, Responsibility of common.....	370
Causeation.....	331
Cause, The specific.....	34
Salmon's "new microbe"—1885.....	52, 53, 55, 57, 60, 63, 70, 74, 76, 237, 239, 241, 245, 247, 298, 391, 395, 396
External.....	12, 17
Internal.....	12, 13
Caution against Pasteur's methods.....	379
Charbon.....	10
Cholera, Asiatic.....	272, 301
Characteristics, Morpho-biological, of germ of swine plague.....	104
Clinical phenomena in Rothlauf-Rouget.....	339
Clinical notes on Pulitz swine—Schutz.....	266
on swine plague in Denmark.....	217
on swine plague in France.....	220, 222
Cold, Influence of.....	31, 324
Coleman, A. R.....	251
Contagion, Swine plague not a.....	17, 18, 23, 25, 26, 27, 28, 267, 274, 285, 319, 358
Definition of.....	274
Contagious diseases more specific in their lesions than infectious.....	300
Conclusions, Salmon's, on Investigations—1885.....	60
Contradictions, Salmon's.....	40, 42, 46, 48, 51, 53, 61, 67, 71, 73, 77, 80, 82, 83, 85, 86, 230, 235, 239, 241, 394
Comparison between Salmon's statements 1884 and 1885.....	61
of Salmon's views on swine plague 1878-1888.....	73
Cornil and Chantmesse...24, 38, 102, 114, 165, 186, 190, 202, 219, 220, 224, 297, 232	
do not find Salmon's hog cholera germ.....	222, 245
Cremation of dead swine.....	368
Darwin.....	346
Definition.....	7, 168
Detmers, H. J., Discoverer of germ of swine plague.....	38, 41, 82, 83, 96, 102, 118, 255, 278, 319, 401

	PAGE
Detmers, H. J., <i>continued</i> —	
letter to author	255
on Salmon's hog cholera germ	247
on lesions	240, 252, 305
on rainfall	30
on entozoa in swine plague	16
"stamped out" by Salmon	39, 43, 96, 359
Differentiation of hog cholera from swine plague—Salmon	76, 83
Between Rose's disease and Salmon's hog cholera	79
Between "Wild-seuche" and swine plague	199, 206
Between Rouget and swine plague	220
Differential diagnosis	14, 19, 176, 198, 220, 330
Diagnosis	176, 177, 342
Disease, Definition of	290
Disposal of sick and dead hogs	368
Duties of the state in prevention	363
Echinorynchus gigas	15, 140
Eggeling, Prof.	190
England, Swine plague in	165, 168
Emphysema-infectiosum—black leg	378
Erysipelas	10, 197, 216, 244, 286, 338
Europe, Swine plague in	165, 166
Experiment, Natural aspiration	24, 143
Experiments, Table of	384
Inoculation	388, 397
Fences, Prevention of infection by	28
Fever, non-recurrent—Salmon	43, 278, 383, 390, 393
Fever, a specific eruptive	168
Feeding, Can swine plague be induced by	307
experiments—rabbits	308
experiments—swine	309, 313
Forgery, Salmon's swine plague (1885) hog cholera (1886-7-8) germ a.	37, 74
111, 216, 222, 233, 243, 246, 298, 331	
Friedberger-Froehner on Wild-seuche	197, 200, 320
Friere, inoculation in yellow fever	344
Gaffky	272, 297, 333
Galen	263
Gelatine, Cultures in	114
Gentilly, France, Swine plague at	186, 220
Genesis of gangreneous pneumonia in swine plague	304
of intestinal lesions in swine plague	315
Geographical distribution	7
Germ of swine plague, Discovery of	11, 24, 82
motile	92, 115
grows on potatoes	81, 92, 114, 115
not spore bearing	77
Goethe	264
Great Britain, Swine plague in	165, 168, 186

	PAGE
Haller.....	264
Harvey.....	264
Hatch, Hon. Wm. H.....	350, 351
Hatch bill.....	8, 314, 351, 353
Haubner.....	11, 14
Helmholtz.....	264, 346
Herschell "Herr College".....	335
Health, Public.....	345, 351
Herring.....	11
History.....	7
of swine plague in England.....	9
Hippocrates.....	263
Hog cholera, Salmon on.....	10, 27, 34, 64, 72, 74, 76, 92, 16, 216, 226, 244, 247, 269
Rose's germ of.....	78, 80, 92
Hogs, Disposal of dead.....	368
Hospital service, U. S. marine.....	350
Hucpe.....	11, 77, 104, 105, 106, 108, 187, 198, 219, 272, 287, 294, 393
on generalization.....	335
Humboldt.....	346
Identity of swine plague with Loeffler-Schutz Schweinesenche.....	14, 190, 199, 207, 224, 244
with Wild-senche.....	14, 190
of the Danish and Loeffler-Schutz germ.....	116
English and American swine plague.....	219
French and American swine plague.....	221
English, Danish, and American disease.....	244
diseases cannot be determined by germs alone.....	224
What constitutes.....	330
of Rose's and Salmon's microbes.....	78
Illustrations.....	402
Immunity acquired.....	119, 172, 374, 382, 386, 387
artificially produced.....	388
Incubation.....	172, 371, 382, 388, 397, 399
Infection, An extra-organismal.....	19, 23, 25, 88, 267, 269, 274, 279, 341
via respiratory tract.....	143
via intestinal tract.....	307
Infectious diseases, What constitutes.....	279
Infiens, Action of.....	282, 300, 302, 322
Inoculation as a preventive of Roug t.....	44, 45, 60, 62, 261, 344, 374, 380, 390
Salmon.....	43, 49, 389, 393
Subcutaneous, uncertain.....	24
Caution as regards.....	377
against Black leg.....	378
Inoculated hogs, necroscopical notes.....	153
rabbits, necroscopical notes.....	192
Institutions, Endowed, never can be true centers of research.....	347
Public, Weakness of.....	347
Intestinal lesions in swine plague.....	307

	PAGE
Intra-vital phenomena in swine plague	320
Investigators, Want of experience by European	299
Jugglery, Mr. Salmon's.....	239, 284
Jenner.....	264, 272, 346, 372
Johnue, Schweineseuche a septicæmia	287
Kitt, Prof.....	114, 115, 188, 198, 219, 244, 302, 378
Kidneys, Embolic obstruction in.....	317
Klein, Dr	8, 10, 38, 41, 45, 93, 96, 165, 169, 219, 302, 318, 323, 399
Criticism of Salmon on.....	41, 43
on Pasteur.....	44
Koch, Prof.....	68, 106, 246, 265, 346
Laboratory, A national.....	349
Requirements of.....	355
Laennec	364
Land, Situation of, as an influence in swine plague	28, 29
Law, Prof	8, 95, 170, 252, 268, 383, 399
first to show the possibility of inoculation in swine plague.....	400
Lesions, External	174, 251
Internal, Walley	177
Gastro-intestinal, Walley.....	180
Glandular, Walley.....	183
in Wild-seuche	189, 198, 201
Roloff's.....	211
Rietsch's.....	223
Cornil-Chantemesse.....	221
Detmers	240, 305
Necroscopical	14, 119, 192, 194, 195, 252, 258, 306, 310, 313, 340
showing swine plague to be a septicæmia	299
pulmonary	303, 319
intestinal	307, 319
feeding experiments in rabbits.....	308
feeding experiments in hogs.....	310, 313
Intestinal, genesis of.....	315
Liver, Cirrhosis of.....	85, 88, 168, 246
Loeffler	9, 44, 52, 114, 115, 118, 167, 190, 192, 197, 200, 202, 219, 222, 2-5, 330
growth of Schweineseuche germ on potatoes.....	84, 199
on septicæmia.....	286
Ludwig	264
Malarial diseases, Definition of	282
Mausfelde, Dr. v.....	289, 291, 292, 295, 297
Man, Not a dollar spent to save him by research.....	344
Marseilles, Swine plague at.....	186, 222, 244
Measles	176
Men, as conveyors of the inficiens.....	27, 28
Micrococci, as the cause of swine plague	37, 38, 42, 43, 45, 46, 47, 51, 52, 53
54, 61, 64, 65, 70, 71, 74, 75, 95, 97, 101, 102, 105, 169, 222, 233, 344, 390	
Microbe of swine plague—Salmon, 1885.....	52, 53, 55, 57, 60, 63, 70, 76, 237, 239
241, 245, 247, 298, 391, 395, 396	

Microbe, <i>continued</i> —	
chronic pneumonia	81
hog cholera, Rose's	78, 80, 86
Miller	264
Morphological identity in germs insufficient to diagnosis	224, 330
Motility of the swine plague germ	83, 115, 221, 224, 241, 244, 332
Nature of swine plague	168, 234, 263, 267
Necroscopical notes	14, 119, 192, 194, 195, 252, 253, 258, 306, 308, 310, 313
Salmon	39, 50, 56, 76, 86
Klein	166, 167
Walley	177
Schutz-Pulitz swine	204, 209
Roloff	211
Cornil-Chantemesse	221
Rietsch	233
inoculated hogs	153
rabbits	192, 194, 196
Wild-seuche	201, 202
Nocard, Prof., quoted by Salmon	41, 42
testifies to the non-existence of Salmon's hog cholera microbe	243, 244
Oedema, enormous	9, 15, 190, 192, 193, 195, 196, 197, 198, 201, 205 206, 208, 244, 336
Osler, Prof.	251
Owners, Responsibility of	28, 358
Pasteur, as the discoverer of the germ of swine plague ...	8, 9, 43, 45, 60, 62, 169, 244
vaccine contra Rouget	44, 45, 60, 62, 264, 344, 374, 380, 390, 391
anti-rabies inoculation	374
Pens, and contents, in causing swine plague	17, 19, 23, 24, 28, 142
Persh, Dr., Salmon's hog cholera germ	247
Pettenkofer, Prof.	264, 272
Phenomena, Intra-vital	320
Clinical, in Rouget	378
Post-mortal, in Rouget	340
Pigeons, Salmon's virus in	393
Pneumonia	145, 176, 251, 271
Prevention	20, 291, 343, 350
duties of owners	368
Salmon on	27, 184, 358, 361
Preventive inoculation	371
Pulmonary lesions in swine plague	303
Genesis of the destructive	304
Purpura hemorrhagica	176
Quarantine, State, for swine on importation	364, 366
Rabbits, Inoculation of	192, 191, 196
Septicæmia of	297
Rabies, Pasteur's prevention of	374
Rats in swine plague	33
Rains, Influences of	29

	PAGE
Reeves, Dr. James E.....	248, 318
Reynault.....	10
Reynal.....	10
Resume of Salmon's views on swine plague, 1878-1888.....	73
of rules of prevention.....	362
Research expensive.....	354
Removal of sick and dead hogs.....	368
Remuneration on the part of state.....	369
Rietsch, Swine plague in France.....11, 38, 102, 115, 165, 186, 219, 222,	232
cannot find Salmon's hog cholera germ.....224, 233, 244	
Rising, Neb., Outbreak at.....	146, 157
Roell, Prof.....	11
Roloff's "Kazige Darm-entzündung".....11, 135, 137, 187, 199, 208, 211, 216,	222, 302, 330, 336
Rothlauf } 9, 12, 44, 60, 72, 191, 107, 200, 202, 206, 216, 220, 244, 338, 340, 374, 380	
Rouget } post-mortal phenomena.....	340
not a contagion.....	340
Rokitansky.....	264
Roberts, Dr. Geo.....	195, 258
Rose, Dr., visits Nebraska.....	78
hog cholera bacterium.....	78, 86
identical with Salmon's.....	78
not identical with Salmon's.....	78, 80
Salmon, micrococci as the cause of swine plague.....37, 38, 42, 43, 45, 49, 50,	
53, 54, 61, 64, 65, 70, 75, 77, 86, 169, 222, 233, 344, 390	
gives up his micrococcus, 1880 to 1885.....	52, 53, 61, 63, 71
Salmon's new microbe, 1885.....	52, 53, 55, 57, 60, 63, 70, 74, 76, 237, 239
241, 245, 247, 298, 391, 395, 396	
two different germs in swine plague.....	37, 64, 67, 77, 222
three different germs in swine plague.....	68, 86
on the identity of Rose's hog cholera germ with his.....	78, 79
on the non-identity of Rose's hog cholera germ with his.....	78, 80
his new swine plague microbe, 1886.....	37, 81, 87, 115, 239, 241
his swine plague germ on potatoes.....	84, 241
how the swine plague germ differs from that of hog cholera.....	241
on the germ of swine plague.....	8, 34
gives no evidence that he ever saw the germ of swine plague.....	88, 91
on the Nebraska germ of hog cholera.....	78, 79, 80
claims Dr. Billings' germ identical with his micrococcus, 1884.....	66
letter to <i>Breeders' Gazette</i>	63
letter to <i>Breeders' Gazette</i> answered.....	66
swine plague but one disease.	34, 35, 37, 42, 52, 53, 55, 60, 61, 62, 65, 73
74, 77, 78, 86, 88, 169, 233, 234, 243, 244, 247, 269, 278, 283, 392	
swine plague two distinct diseases.....	34, 37, 74, 75, 76, 77, 78, 81, 161, 164
166, 187, 216, 226, 283, 323	
contradictions.....	27, 40, 42, 46, 48, 51, 53, 61, 67, 71, 73, 76, 78, 81, 82,
83, 86, 230, 235, 239, 242, 391, 396	

Salmon, *continued*—

unfounded statements by.....	67, 72, 21, 307
views compared, 1878-1888.....	73
views compared, 1880-84 with those of 1885.....	61
conclusions, 1885.....	60
sends to Europe for testimony against himself.....	243
says swine plague is a contagion.....	20, 24, 27, 37, 278, 285, 358
says swine plague is not a contagion.....	296
"stamps out" Dr. Detmers.....	39, 43
on the nature of swine plague.....	35, 36, 37, 4, 268, 269, 278, 285, 296
criticises Dr. Klein.....	41, 44, 46
quotes Pasteur.....	43, 62, 392
"how to prevent hog cholera".....	20, 27, 43, 278
reliable virus.....	43, 49, 50, 389, 390, 393
cirrhosis hepatis in swine plague.....	85, 88, 246
belief.....	392
Selander.....	11, 190, 218
Septicæmia, swine plague, a.....	17, 19, 23, 25, 155, 192, 197, 198, 207, 267, 271, 282
283, 285, 286, 295, 298, 319, 323, 376, 381	
definition of.....	288
a specific disease.....	289
surgical.....	294, 298
an extra-organismal, defended.....	295
intra-organismal.....	296, 298
Service, U. S. Marine Hospital.....	350
Schutz differentiates Schweineseuche from Rouget.....	9, 44, 52
on Roloff's lesions.....	14
on Pasteur's vaccine contra Rouget.....	44
the germ of Schweineseuche.....	72, 82, 83, 187, 199
really discovers swine plague.....	9, 199, 200, 204, 207, 209, 216
on pneumonia.....	285
contradicts himself.....	286, 287
erroneous pathology.....	288
want of practical experience.....	299
"Ueberrashung".....	330, 335
Schweineseuche.....	9, 11, 14, 18, 72, 81, 83, 146, 190, 199, 201, 202, 217, 219, 222
243, 244, 284, 285, 320, 333	
a septicæmia—Loeffler.....	286
a septicæmia—Schutz.....	286
a septicæmia—Hueppe.....	287
Schottelius—Lydtin on Rouget inoculation.....	44, 380
Sclerosis hepatis.....	85
Smith, Dr. Theobald.....	64, 65, 68, 87
Spores, Not developed.....	77
Southern cattle plague.....	281, 369
Statistics.....	8, 10, 35
Strebel.....	379
Streamis extend swine plague.....	27, 31

	PAGE.
State, Duties of the.....	363, 369
Sutton, Dr. G.....	10
Swine fever.....	9, 10, 170, 171, 244
Swine plague in Canada.....	8, 251
in Germany.....	8, 11, 14, 165, 200, 204, 209, 211, 217
in France.....	11, 102, 165, 186, 190, 219, 220, 222, 243
in Sweden and Denmark.....	11, 15, 165, 187, 190, 217, 244
in Russia.....	11
a septicæmia as shown by its lesions.....	299
causes of.....	12
internal causes of.....	12, 13
external causes of.....	12, 17
its specific cause.....	12, 13, 34, 53, 81, 87
Roloff's lesions in.....	11, 14, 135, 137, 187, 199, 208, 211, 216, 222, 302, 330, 336
Echinorynchi in.....	15, 140
diseased swine as a cause of.....	17, 18, 143
not a contagion.....	17, 18, 21, 23, 25, 26, 27, 28, 267, 269, 274, 319
earth and refuse as a cause of.....	19, 23, 24, 28, 142, 280, 360
at Valparaiso, Neb.....	23, 142
at Rising, Neb.....	146, 157
at state farm.....	25
effect of seasons, etc.....	18, 21, 24, 26, 29, 143, 324, 398, 399
effect of seasons, etc.—Detmers.....	30
effect of running streams.....	17, 27, 31
effect of stock cars.....	17, 32
effect of rats.....	33
SWINE PLAGUE ONE DISEASE.....	34, 35, 37, 42, 52, 53, 60, 65, 73, 78, 81, 86, 93
102, 108, 161, 163, 166, 171, 195, 216, 226, 233, 234, 240, 244, 247, 251	
253, 283, 318	
nature of.....	35, 36, 37, 168, 263, 279, 282, 318
Salmon's new microbe, 1885.....	53, 55, 57, 60, 63, 70, 76
Salmon's conclusions, 1885.....	60
Salmon's conclusions, 1880-1885.....	61
two different germs in—Salmon.....	64, 67
three different germs in—Salmon.....	68, 78, 79, 86
germs not spore bearing.....	75, 77
a chronic pneumonia—Salmon.....	76, 86, 87, 162
Rose's microbe.....	78, 79, 86
Detmers first discovers the germ.....	38, 82
Salmon's reliable vaccine.....	43
symptoms.....	173, 252
induced by feeding.....	307
author's conclusions.....	318
what is it?.....	318
diagnosis.....	342
prevention.....	342
Testimony of the non-existence of Salmon's hog cholera germ.....	243
Temperature.....	323

	PAGE
Treatment.....	184
Typhus abdominalis.....	10, 168, 272, 293
Uremia.....	296
Uticaria.....	176
Vaccine—Salmon's reliable.....	43, 49, 50, 389, 393
Vaccine, contra Rouget.....	62, 344, 374, 380, 390
Vaccination.....	372
Valparaiso, Neb.....	23, 154
Variola.....	177
Variolation.....	372
Vesalius.....	264
Virchow.....	264, 332
Virus.....	27, 172
Walley, Prof.....	120, 137, 168, 317, 383
Welch, Prof.....	231, 246, 247
Wealth, Public.....	345, 351
What then is swine plague?.....	318
"Wild-seuche".....	9, 12, 14, 15, 105, 107, 120, 188, 190, 195, 197, 198, 201, 203, 208 217, 219, 244, 272, 285, 287, 294, 301, 333
Wing's letter on swine plague at College Farm.....	25, 397

SWINE PLAGUE—PLATE I.



FIG. 1.
Salmon's Micrococcus, U. S. Agricultural Report, 1880.

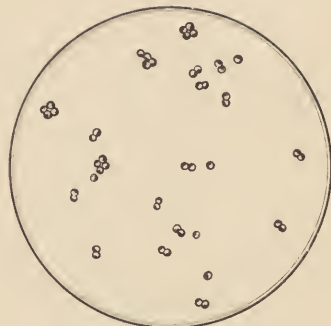


FIG. 2.
Salmon's Micrococcus, U. S. Agricultural Report, 1884.

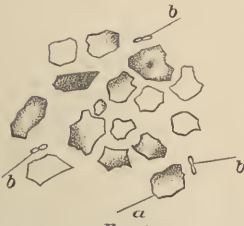


FIG. 3.
Salmon's "New Swine Plague Microbe,"
U. S. Agricultural Report, 1885.



FIG. 4.
Salmon's "Hog Cholera Microbe," (same as Fig. 3)
U. S. Agricultural Report, 1886.

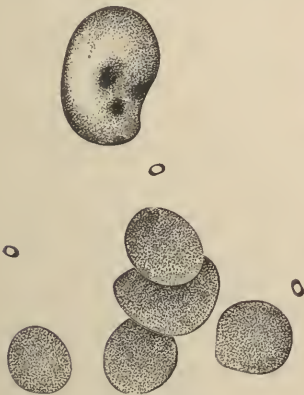


FIG. 5.
Salmon's Swine Plague Germ, U. S. Agricultural Report, 1886.

SWINE PLAGUE—PLATE II.

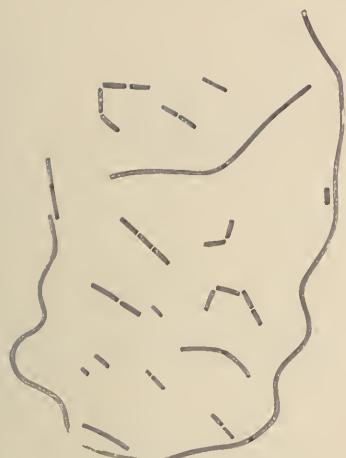


FIG. 1.
Bacillus Subtilis.—Klein. Micro-organisms
and Disease. Fig. 39, p. 75.



FIG. 2.
Bacillus Subtilis developing spores.—
Klein, l. c. Fig. 40, p. 76.



FIG. 5.
Hanging drop culture of the Germ of
Swine Plague.



FIG. 3.
Klein. "Fig. 63," p. 87.
Bacilli of Swine Plague.



FIG. 4.
Klein. Fig. 64, p. 97.
Bacilli of Swine Plague.

SWINE PLAGUE.—PLATE III.

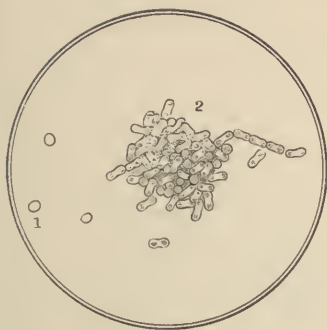


FIG. 1.
From Dr. Detmers' illustration of the
Germ of Swine Plague, in *American
Naturalist*, vol. 16.



FIG. 2.
From a recent photograph by
Dr. Detmers, 1888.



FIG. 3.
The true Swine Plague Germ. Mature form.



FIG. 4.
Diagrammatic. Illustrating the phases
of development in a hanging drop
culture of the Germ of Swine
Plague.



FIG. 5.
Bacterium of Rabbit Septicæmia,
according to Baumgarten.

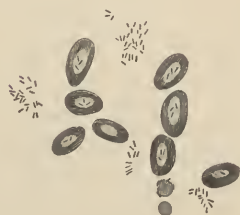
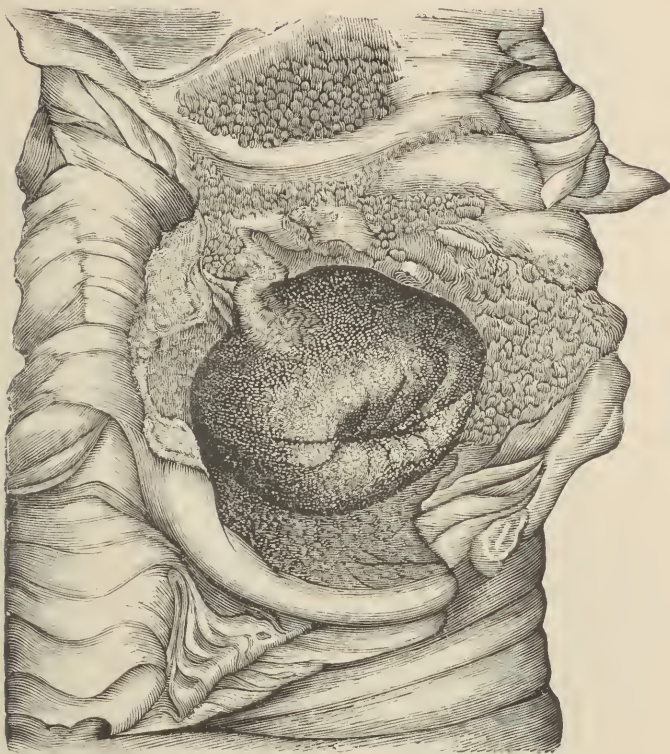


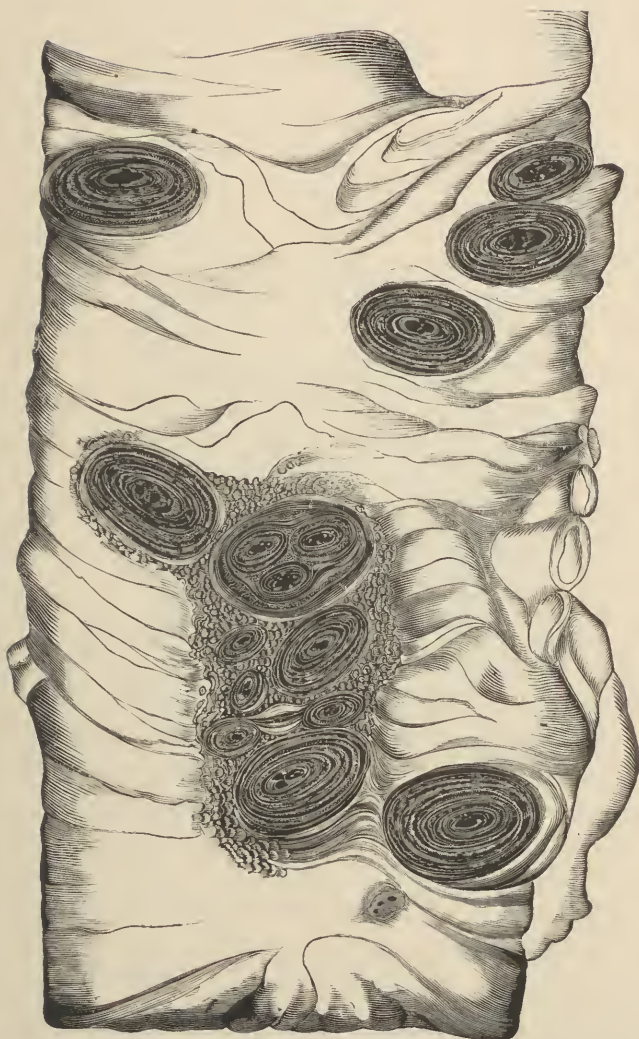
FIG. 6.
Bacillus of Rouget (Schütz)
from blood of inoculated
pigeon.

SWINE PLAGUE.—PLATE IV.



Cæcum of Hog with indurated ileo-cæcal valve, as described by Roloff, in
Germany, in 1875.
See also Plate IV.
See Autopsy IX., p. 137.

SWINE PLAGUE.—PLATE V.



Cæcum of Hog with indurated neoplastic products of embolic origin, as described by Roloff, in Germany, in 1875.
See autopsy IX., p. 137.

SWINE PLAGUE.—PLATE VI.



Cæcum of inoculated pig, showing same condition
of ileo-cæcal valve and surrounding tissues, as
illustrated in previous Plate.
Autopsy XXX., p. 159.

SWINE PLAGUE.—PLATE VII.



Chronic neoplastic indurations in caecum, due to embolism.
Inoculated hog No. 45. Autopsy XXXI., p. 163.

SWINE PLAGUE.—PLATE VIII.



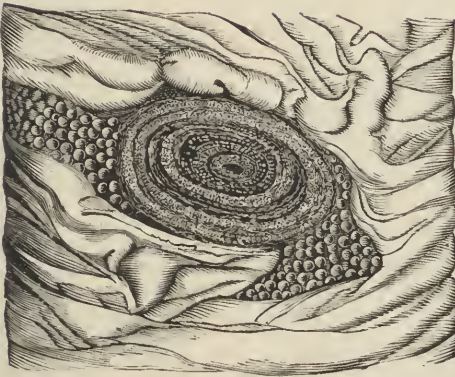
Chronic neoplastic indurations, due to external embolism
in walls of colon, inoculated.
Swine Plague, Fig 45. Autopsy XXXI., p. 163.

SWINE PLAGUE.—PLATE IX.



Acute follicular ulceration, due to immediate embolic
obstruction of circulation in caecum.
Autopsy II., p. 1:5.

SWINE PLAGUE.—PLATE X.



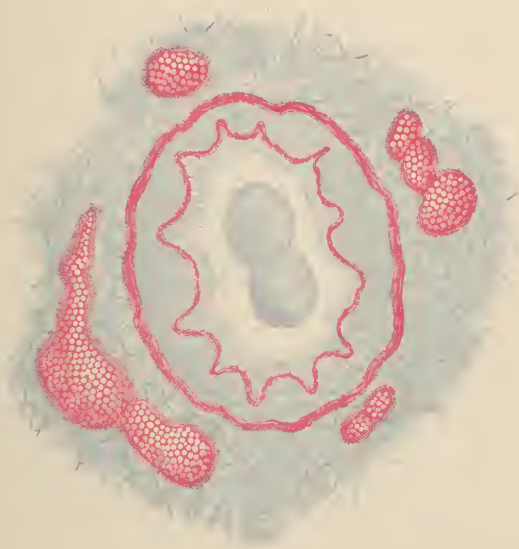
Neoplastic growth in consequence of protracted
embolic obstruction of the circulation in the
walls of the intestine.
See Autopsy VII., p. 134.

SWINE PLAGUE.—PLATE XI.



Acute caseous follicular ulceration and peri-induration
in cecum.

SWINE PLAGUE.—PLATE XII.



Coagulation, obstruction of the circulation in the lungs,
illustrating the first stage of destructive pneumonia
in Swine Plague.

3272





SF B598sa 1888

62310600R



NLM 05093304 3

NATIONAL LIBRARY OF MEDICINE